

MEDICINE

PART

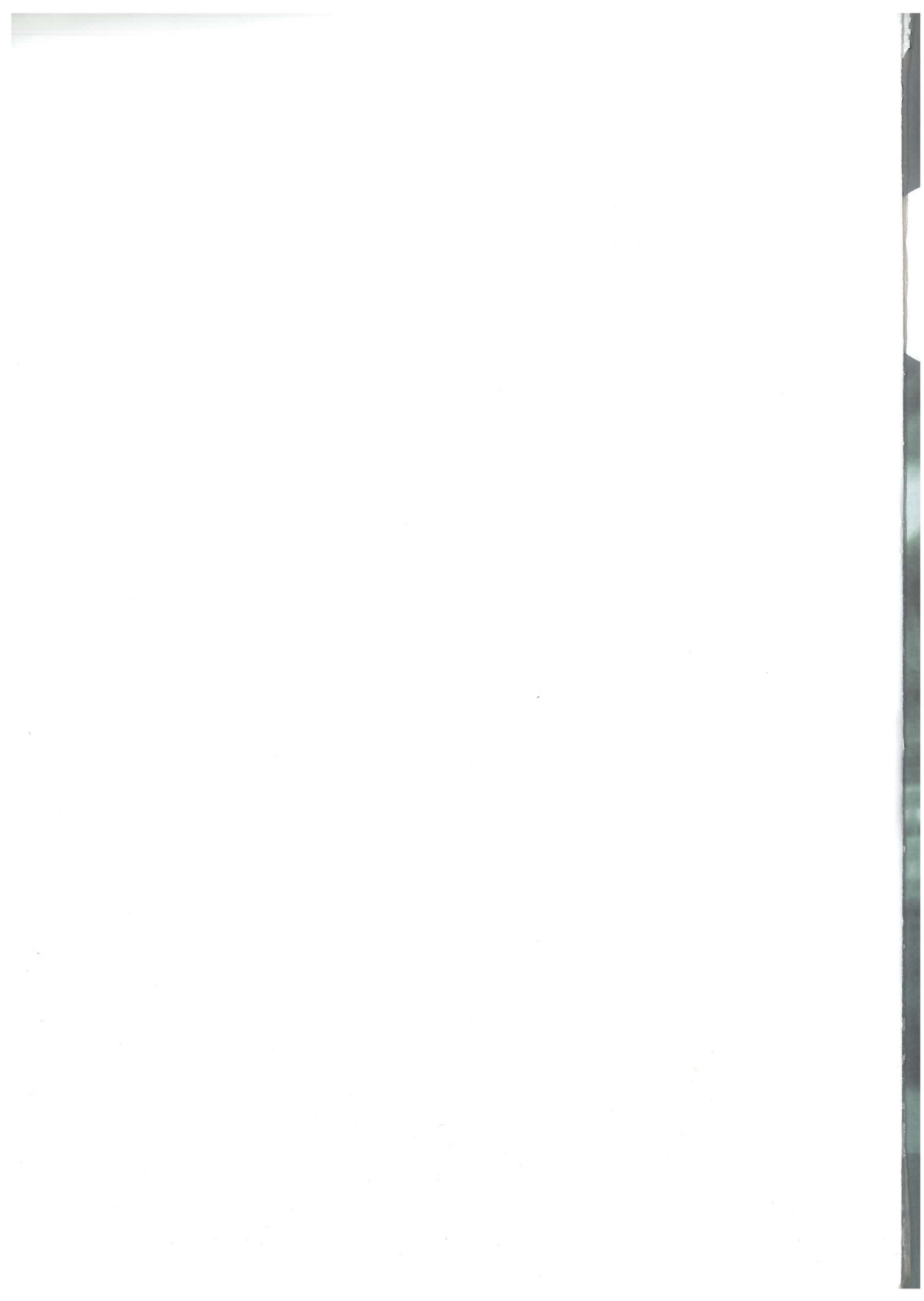
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# OPHTHALMOLOGY



IRIS  
ASPECT™

WAEEL SALAH



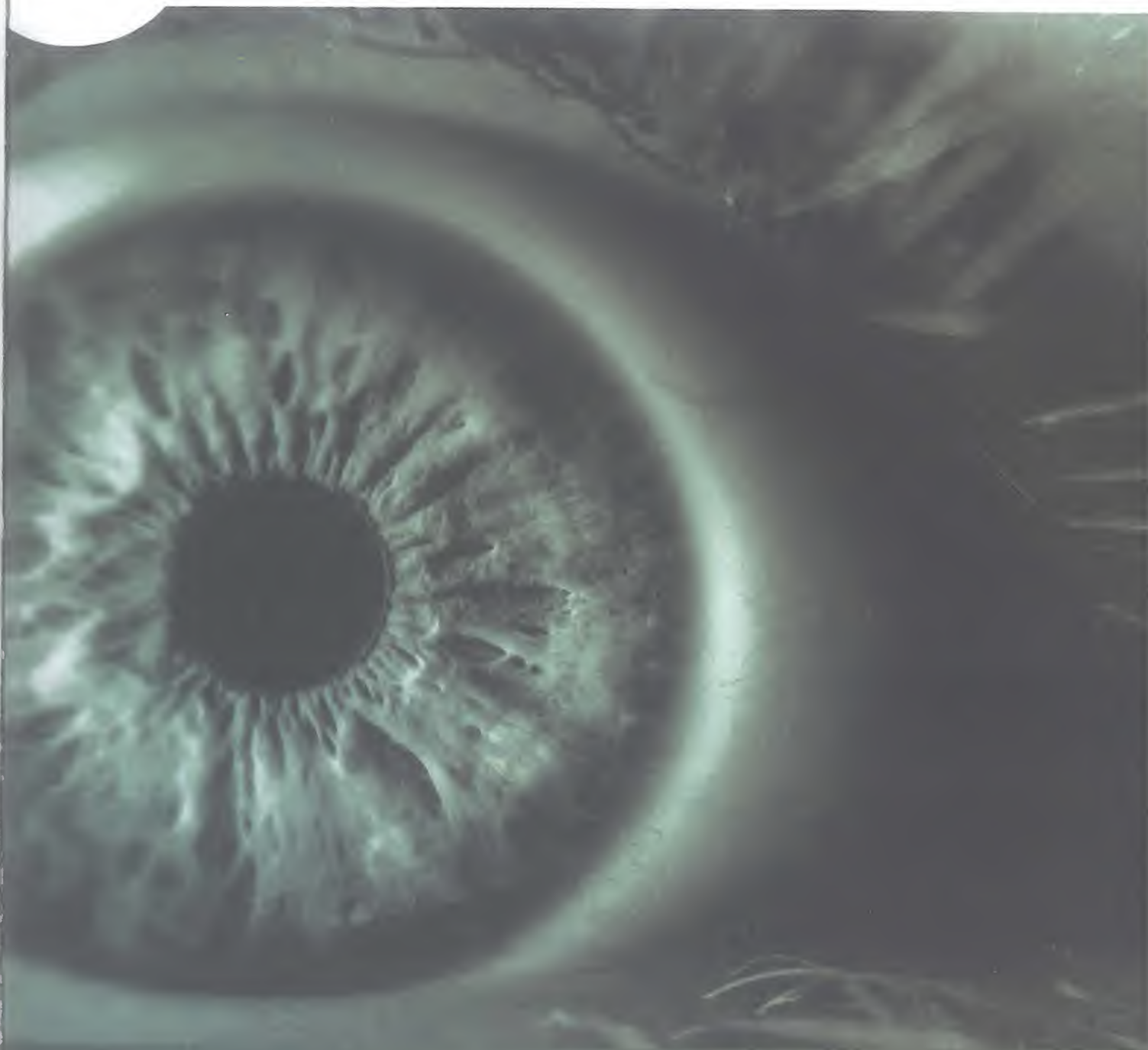


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1

# OPHTHALMOLOGY



IRIS  
ASPECT

WAEEL SALAH

*Edited by :*

*Dr . Hossam Nasr*

رقم الإيداع

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## Index

The Eye ball .....	1
Eyelids .....	3
The Lacrimal apparatus .....	27
Conjunctiva .....	38
Cornea .....	62
Sclera .....	93
Lens .....	98
Uveal Tract .....	124

# The Eye ball

✚ The eye ball is a sphere with a diameter of **24 mm** (about 1 inch).

## 1) Outer coat is a fibrous layer and is formed of:

- a) **Cornea:** Anterior  $\frac{1}{6}$  is transparent & avascular.
- b) **Sclera:** Posterior  $\frac{5}{6}$  is opaque & white in color.

## 2) Middle coat: Is called uveal tract pigmented coat, vascular coat & nutritive coat.

It is divided into:

- a) **Iris (Anteriorly).**
- b) **Ciliary body (middle part).**
- c) **Choroid (Posteriorly).**

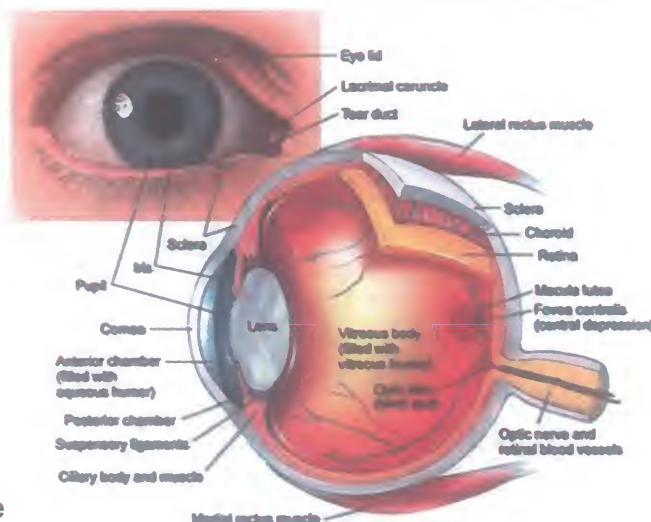
## 3) Inner coat:

✚ **Neuro-sensitive layer or the retina.**

✚ The retina is capable of transforming photon energy into electrical impulses which is transmitted through optic nerve and visual pathway to the visual areas in the occipital cortex.

✚ Crystalline lens is present posterior to the iris suspended in place by Zonules (suspensory ligaments) which is attached to the inner surface of the ciliary body.

✚ Cornea & Crystalline lens are **the refractive surfaces of the eye.**



**The cavity of the eye is divided into:**

## 1) Posterior cavity (vitreous chamber):

Lies between the lens & the retina and is occupied by the vitreous gel.

## 2) Anterior cavity:

✚ Lies between the lens & the cornea. This cavity is subdivided by the iris into:

- a) **Anterior chamber** lies between the cornea and the iris.
- b) **Posterior chamber** lies between the iris & the lens.

✚ The anterior & posterior chambers communicate through the pupil.

✚ The anterior & posterior chambers are filled with a clear fluid called aqueous humor,

✚ The aqueous humor is secreted by the ciliary body inside the posterior chamber then passes through the pupil to reach anterior chamber where it's drained through the angle of the anterior chamber.

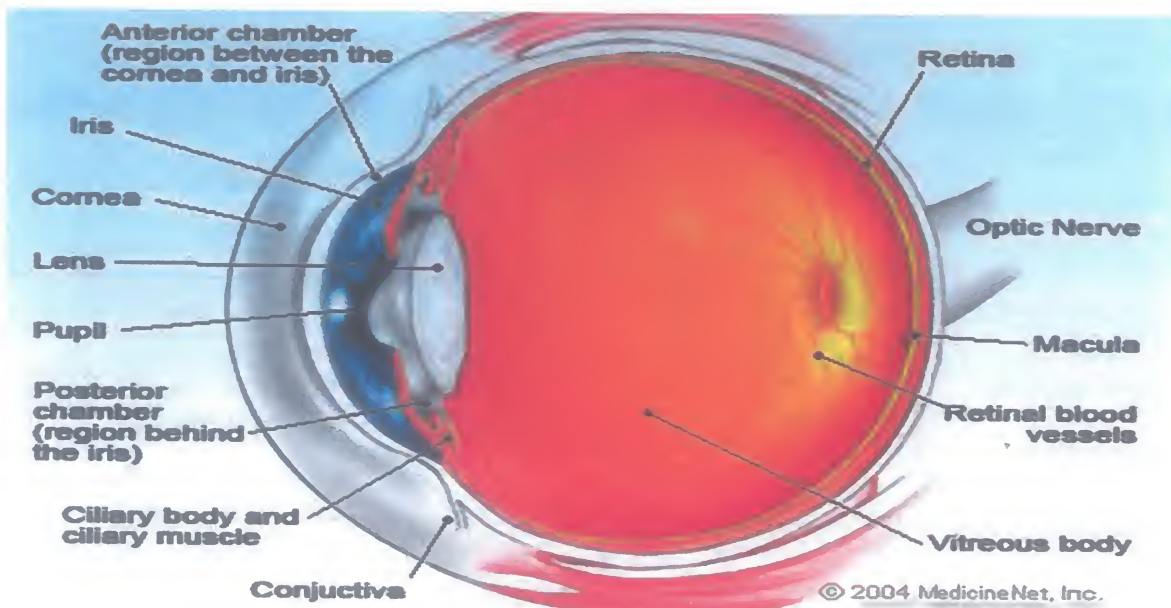
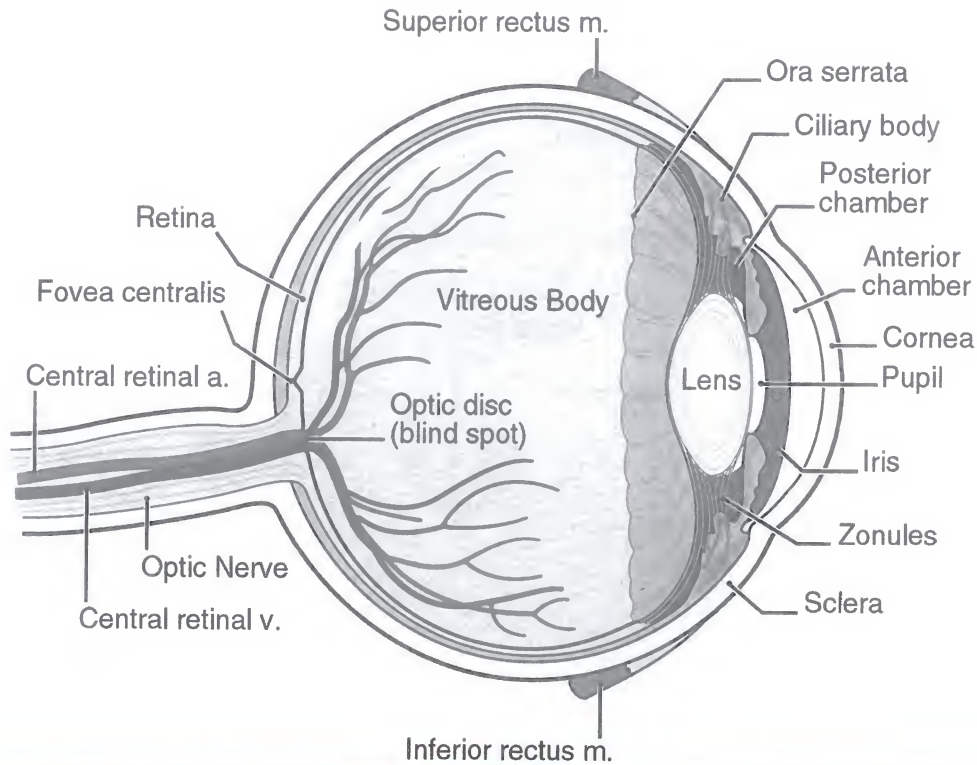
✚ Intraocular pressure ranges between 10 - 22 mmHg.



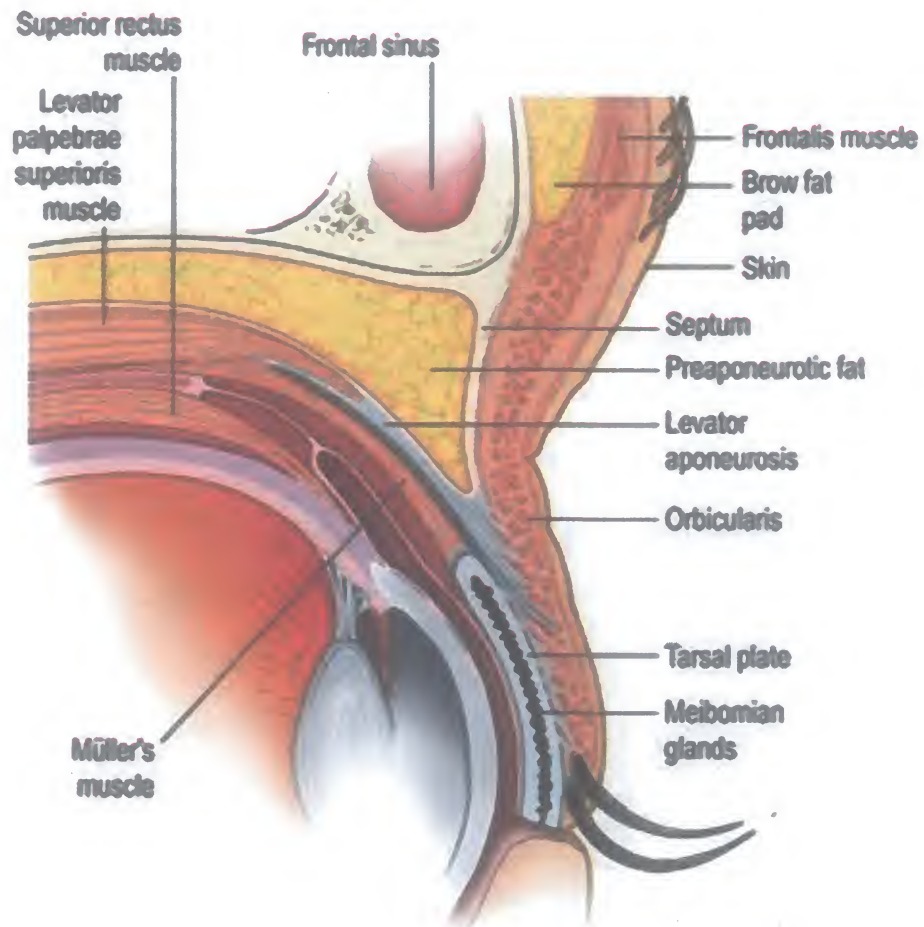
## Ocular adnexia

Is the accessory structures related to the eyeball, they are:

- a) Eyelids.
- b) Conjunctiva,
- c) Lacrimal apparatus.
- d) Extra-ocular muscles.
- e) Orbit.



# EyeLids



# Eyelid

❖ The eye lids are two movable mucocutaneous folds, which acts as shutters.

## 1) THE UPPER LID:

- a) Extends to the eye brow.
- b) Covers the upper 1/6 of the cornea.
- c) Shows a crease when it is raised.

## 2) THE LOWER LID:

- a) Passes into the cheek without a demarcation line.
- b) Rest at the level of the lower limbus.

## 3) THE PALPEBRAL FISSURE:

✚ It is the space between the lid margins when the lids are open:

- a) **Shape:** Elliptical.
- b) **Angle:** Canthi (medial canthus “rounded” and lateral canthus “acute”).
- c) **Length:** About 30mm.
- d) **Width:** In males ranges from 7-10mm  
In females ranges from 8-12mm (average 10mm).

## 4) MINUTE ANATOMY:

✚ A sagittal section in the upper eyelid shows:

- a) Skin.
- b) Subcutaneous layer.
- c) Striated muscle layer.
- d) Sub-muscular layer.
- e) Fibrous layer.
- f) Palpebral conjunctiva.

### a) Skin:

Thinnest skin in the body, less than 1 mm in thickness.

### b) Subcutaneous layer:

- ❖ Very loose.
- ❖ Rich in elastic fibers.
- ❖ Almost devoid of fat.

### c) Striated muscle layer: it includes:

#### 1- Orbicularis oculi muscle:

It is the sphincter of the eye lids and consists of:

##### a) Orbital portion:

**Origin:** medial palpebral ligament

**Insertion:** medial palpebral ligament after it makes complete circle around orbital margins

**Action:** forcible closure of eyelids.

##### b) Palpebral portion:

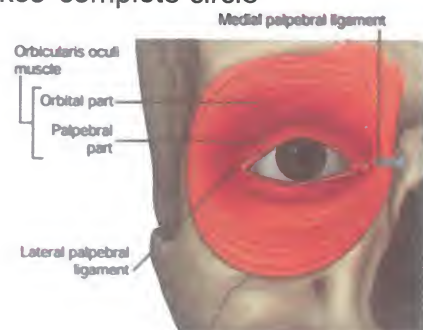
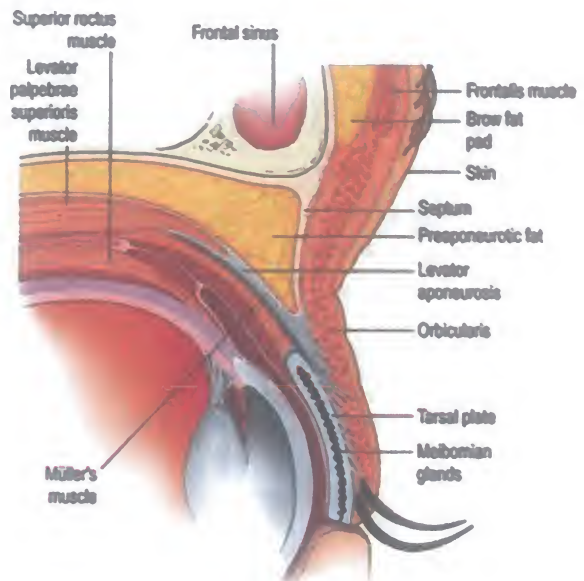
**Origin:** From medial palpebral ligament.

**Insertion:** Lateral palpebral ligament.

**Parts:** Pre-septal & Pre-tarsal.

**Action:** 1- Gentle closure of the eyelid.

2- Support lower lid against gravity.





c) **Marginal fibers (muscle of Riolen):** This muscle is close to lid margin.

d) **Lacrimal portion (Horner's muscle):**

**Origin:** Posterior lacrimal crest.

**Insertion:** Lacrimal fascia.

**Action:** Pulls on lacrimal sac, Creates -ve pressure inside the sac, withdraw tears from the canaliculi.

**Nerve supply:** Facial nerve (7<sup>th</sup> n).

**Paralysis:** Lagophthalmos – Epiphora – Paralytic ectropion.

## 2- **Levator palpebrae superioris:**

**Origin:** Orbital apex above and in front of the optic foramen (lesser wing of sphenoid).

**Course:** Flat ribbon like muscle, runs above superior rectus close to the roof of the orbit, one centimeter before orbital septum, it fans out into aponeurosis, which extends the whole width of the eyelid.

**Insertion:**

- a) Upper fornix.
- b) Upper part of the anterior surface of the tarsus (main insertion).
- c) Skin forming a crease.
- d) Medial horn to medial palpable ligament.
- e) Lateral horn to lateral palpebral

**Action:** Elevation of upper lid.

**Nerve supply:** Oculo-motor nerve.

**Paralysis:** Ptosis.

### d) **Sub-muscular layer.**

### e) **Fibrous layer:**

**1- Tarsus:** It is formed condensed fibrous tissue resembling cartilage.

**Attachment:**

- ❖ Medially and laterally to the palpebral ligaments,
- ❖ Superiorly to Muller and levator muscles.

**Dimensions:**

- ❖ **Horizontally:** 27 – 30 mm.
- ❖ **Vertically:** upper tarsus 10mm & lower tarsus 3-4mm.
- ❖ **Thickness:** 1 mm.

**N.B. 1: Embedded inside the tarsus vertically arranged meibomian glands (20 - 30 in the upper lid & 10 – 15 glands in the lower lid)**

**N.B. 2: Meibomian glands open each by a single duct on the lid margin to secrete outer oily layer of the tear film**

**2- Orbital septum:** Thin fibrous membrane extending from tarsus to the orbital margin.

### f) **Palpebral conjunctiva:**

- ✚ It is thin, vascular and is firmly adherent to the tarsus.
- ✚ Two millimeters above lid margin, it shows a horizontal groove called sulcus subtarsalis.



### Non striated muscle layer (Muller's muscle):

#### **Origin:**

Arises together with levator muscle at the orbital apex passing below levator muscle.

**Insertion:** Upper edge of the tarsus.

**Action:** Helps in elevation of upper eye lid.

**Nerve supply:** Superior cervical sympathetic ganglion.

**Paralysis:** Horner's syndrome (ptosis - miosis - anhydrosis - enophthalmos).

## Lid Margin

- ❖ It is the free margin of the lid.
- ❖ It is 2 – 3 mm. broad.
- ❖ The lid margin is divided by the lacrimal papilla into:

1- Lacrimal portion: From lacrimal papilla to the medial canthus.

2- Ciliary portion: From lacrimal papilla to the lateral canthus & **show**:

- a) Ant. Border: Rounded and carries eyelashes (150 in upper lid and 75 in lower lid)
- b) Grey line: Lies in front of ducts of Meibomian glands. Its incision splits the lid into:
  - **Posterior portion**: containing the tarsal plate and conjunctiva.
  - **Anterior portion**: containing orbicularis oculi muscle, skin, hair follicle.
- c) White line: Opposite to the openings of the ducts of Meibomian glands.
- d) Post. Border: Sharp (for conduction of tears to puncti) and in contact with the eye.

## Glands of the eye lids

1) Meibomian Glands: (which secrete oily layer of tear film):

- a) Prevents overflow of tears over the lid margin.
- b) Retard evaporation of tear film.
- c) Lubricate movement of eye lid along the globe.

2) Zeis glands: Modified sebaceous glands related to lashes (for its lubrication).

3) Moll's glands: Modified several glands related to lashes (for its lubrication).

## Blood Supply

1) Arteries:

- a) Medial palpebral arteries: (branches from ophthalmic artery), which **divides into**:  
Superior and inferior branches for the upper and lower lids.
- b) Lateral palpebral arteries: (branches from lacrimal artery), which **divides into**:  
Superior and Inferior branches.

- ❖ These vessels anastomose to form **2 tarsal arcades in the upper eyelid** (one arcade near lid margin and the other arcade at the upper border of the tarsus) and **one tarsal arcade only in the lower lid**.
- ❖ These tarsal arcades receive anastomotic branches **from**:
  - 1- Superficial temporal arteries.
  - 2- Transverse facial arteries.
  - 3- Infraorbital arteries.

2) **Veins:** Arranged in pretarsal & postarsal plexuses draining into **ophthalmic vein**.

3) **Lymphatic drainage:**

a) Lateral part ( $\frac{2}{3}$ ) of the lid is drained to the **pre-auricular & parotid L.N.**

b) Medial part ( $\frac{1}{3}$ ) of the lid is drained to **submaxillary L.Ns.**

(1 & 2 are drained to the **deep cervical L.N.**)

4) **Nerve supply:**

a) **Motor:**

- Orbicularis → Facial nerve
- Levator → Oculo-motor
- Muller's → Sympathetic fibers

b) **Sensory:**

1- **Upper lid:**

- Supra orbital "Mainly"
- Infra trochlear & Supra trochlear "Medial side"
- Lacrimal branch of ophthalmic "lateral side"

2- **Lower lid:**

**Infraorbital** with minimal overlap near the angle by the **infra trochlear & lacrimal N.**

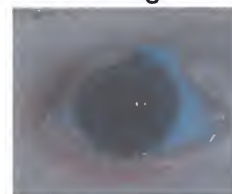
## Congenital anomalies of Eye lids

**1- Epicanthus:**

Vertical concave skin fold extending between the upper and the lower lid margins medially

**2- Coloboma of lid:**

Congenital defect (triangular defect) of lid margin.



**3- Blepharophimosis:**

Generalized narrowing of palpebral fissure and frequently associated with epicanthus & ptosis.

**4- Distichiasis:**

Extra row of lashes.



**5- Congenital Ptosis.**

**6- Congenital Entropion and Ectropion.**



**Epicanthus**



**Coloboma of lid**

## Lid edema

- 1) **Traumatic**: due to injuries or insect sting.
- 2) **Inflammatory**: it is an active edema
- 3) **Non-inflammatory**: Either Allergic (by medication) or Passive (by systemic disease).

## Inflammations of the eye lid

- 1) Inflammations of lid proper → **Lid abscess**.
- 2) Inflammations of lid margin → **Blepharitis**.
- 3) Inflammations of lid glands → **(see later)**.

## Lid Abscess

**Definition**: Localized suppurative inflammation of the lid.

**Clinical picture**: Painful, red, hot, tender swelling.

**Treatment**: Antibiotics (local and systemic) or surgical drainage.

## Blepharitis

**Definition**: Chronic inflammation of the lid margin.

**Predisposing factor**:

- |                |                                    |                |
|----------------|------------------------------------|----------------|
| ❖ Old age      | ❖ Diabetes                         | ❖ Malnutrition |
| ❖ Avitaminosis | ❖ Uncontrolled error of refraction |                |

**Types**:

- |                            |                                     |
|----------------------------|-------------------------------------|
| 1) Squamous blepharitis.   | 3) Parasitic blepharitis            |
| 2) Ulcerative blepharitis. | 4) Angular blepharo-conjunctivitis. |

## 1 - Squamous blepharitis

**Etiology**:

- ❖ The condition is essentially metabolic being a manifestation of **seborrhea**.  
(Dandruff of the scalp is often present).
- ✚ **Infection** by weak organism which splits **Zeis gland** secretion into irritant fatty acids.

**Clinically**:

- 1) Small, **white scales** are present between the lashes.
- 2) Removal of the scales reveals a **hyperemic** lid margin.

**Complications**: Ptylosis (thickening & hypertrophy of lid margin).



### **Treatment:**

- 1) General treatment of seborrhea
- 2) Remove scales by a piece of cotton wetted by 3% **sodium bicarbonate** or by using baby shampoo.
- 3) Apply **antibiotic ointment** into the lid margin.
- 4) **Steroid eye ointment.**



## 2- Ulcerative Blepharitis

### **Etiology:**

- 1) Predisposing factors (see before).
- 2) **Exciting cause:** Infection of the lid margin with **staphylococcus aureus**.

### **Clinical picture:**

**Symptoms:** Burning sensation, lacrimation, frequent falling of lashes and discharge

**Signs:** yellow crusts glue the lashes together, and when removed they leave minute ulcers at the lid margin which bleed easily

**D.D.:** Dried discharge in cases of conjunctivitis and squamous blepharitis.

### **Sequelae:**

- 1) Chronic conjunctivitis.
- 2) Marginal corneal ulcer.
- 3) Styes.
- 4) **Madarosis** due to destruction of the hair follicles  
(area in which lashes are Absent.)
- 5) Trichiasis.
- 6) **Ptylosis:** thickening and hypertrophy of the lid margin.
- 7) Eczema.
- 8) Ectropion.
- 9) **Epiphora:** due to destruction of the sharp post. lid margin, this will initiate the vicious circle of:





### **Treatment:**

#### **1) General treatment:**

- a) Improve general health.
- b) **Control diabetes** if present.
- c) **Correct error of refraction.**

#### **2) Local treatment:**

- a) Remove of crusts: With soap & water or **3 % sodium bicarbonate** lotion.
- b) Apply **antibiotic ointment** (according to sensitivity) & rub it into the margin because the organisms are **hidden** in the hair follicles.
- c) Continue the same treatment for **2 weeks** after apparent cure.
- d) **Epilate** any maldirected lash (No electrolysis).

## 3- Angular blepharitis

### **Etiology:**

Chronic infection of the lid margin by **Morax Axenfeld diplobacillus** (which is Gram negative organism secreting proteolytic enzyme, causing epithelial maceration).

### **Clinical picture:**

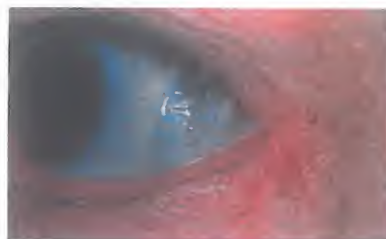
**Symptoms:** Itching + Discharge.

**Signs:** Inner and outer canthi show **maceration** - Conjunctival **redness and discharge.**

**Complications:** Corneal ulcer & Ankyloblepharon (adhesions between the two lid margins)

**Treatment:** As organism is **sensitive to Terramycin** & proteolytic enzyme could be inactivated by **zinc**, so patients **are given:**

- 1) **Zinc sulphate drops**  $\frac{1}{2}$  % t.d.s.
- 2) **Terramycin ointment**  $\frac{1}{2}$  % t.d.s.
- 3) **Gentian Violet 1% paint** (for the macerated skin).



### **Why Angular?**

Due to relative tear deficiency at the angle (i.e. deficiency of lysozyme) which can protect the conjunctiva against bacteria.

## 4- Parasitic blepharitis

**Etiology:** It is due to infestation with **Phthirus pubis** (the pubic louse) & rarely by the head louse

The cilia are covered with nits which are **glued** to lashes by **alkaline cement.**

### **Treatment:**

- 1) **Dilute acetic acid 2%** is used to loosen the nits.  
It dissolves the cement sub. which glues nits to the cilia.
- 2) **Yellow oxide of mercury ointment 1%** applied to the lid.  
It destroys the larvae, continue for 3 weeks.
- 3) **Cutting the lashes** may facilitate the treatment.



# Inflammation of the glands of the lid

## 1 - Sty (Hordeolum externum)

### **Definition:**

Acute suppurative inflammation of Zeis gland and the lash follicle, forming a small abscess.

### **Etiology:**

- ❖ Infection of a Zeis gland by **staphylococcus aureus**.
- ❖ **Predisposing factors:** Diabetes, poor general resistance, errors of refraction & ulcerative blepharitis.

### **Clinical picture:**

#### Symptoms:

- 1) Swelling of the lid.
- 2) Severe pain, first dull then throbbing.

#### Signs: Diffuse red swelling:

- 1) Related to a lash
- 2) Close to the lid margin
- 3) Points on the skin side.

#### Complications:

- 1) Madarosis.
- 2) Cavernous sinus thrombosis.
- 3) Orbital cellulitis.

#### D.D.: Hordeolum internum.

### **TREATMENT:**

- 1) Hot fomentations.
- 2) Local antibiotic drops and ointment.
- 3) Systemic antibiotics.
- 4) When pointing occurs, the pus must be **evacuated** by:
  - a) Epilation of the related lash.
  - b) Horizontal incision.
- 5) For recurrent cases: **correct the underlying cause**





## 2 - Hordeolum internum

### Definition:

Acute suppurative inflammation of the meibomian gland caused by Staph. Aureus.

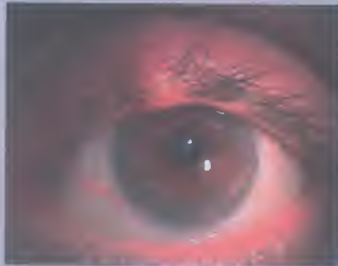

- ✚ It may be primary or it may occur on top of a chronic inflammation of the meibomian gland (chalazion).

### Clinical picture:

**Symptoms:** (As sty).

**Signs:** Diffuse lid swelling which when points appears as yellowish spot of pus through conj. when lid is everted.

**D.D.:** Hordeolum internum should be differentiated from Hordeolum externum (stye).

	Hordeolum externum (Stye)	Hordeolum internum
<b>Site</b>	At the <b>lid margin</b> related to a lash	In <b>tarsus</b> deep to the orbicularis muscle
<b>Inflammatory signs</b>	Mild	Marked
<b>Swelling</b>	Related to a <b>lash</b>	<b>Yellowish spot of pus</b> is seen shining through the <b>palpebral conjunctiva</b>
<b>Contraction of orbicularis</b>	Not affected	Diminished
<b>Treatment</b>	<ul style="list-style-type: none"> <li>❖ Antibiotics</li> <li>❖ Horizontal incision to evacuate the pus</li> </ul>	<ul style="list-style-type: none"> <li>❖ Antibiotics</li> <li>❖ <b>Vertical conjunctival incision</b> or a <b>horizontal skin incision</b> &amp; evacuate the pus</li> </ul>
		

# Chalazion (Cyst)

**Definition:** It is a chronic non-specific **inflammatory granuloma** of a meibomian gland.

**Etiology:** Unknown and may be **produced by:**

- 1) **Chronic irritation** by a low virulence organism.
- 2) **Retained contents of the gland** following obstruction of its duct by:
  - a) Proliferation of epithelium (vitamin A deficiency).
  - b) Dry secretions.



**N.B.1:** The retained secretion is irritant and excites a **granulomatous reaction**.

**N.B.2:** The granuloma contains many giant cells.

**Clinical picture:**

## Symptoms:

- 1) **Painless swelling** of a **long duration** felt under the skin of the lids.
- 2) Pain occurs only when it becomes **infected (acute chalazion)**.

## Signs:

- 1) A slowly growing **painless swelling of the tarsus**.
- 2) If the lid is everted, the conjunctiva is seen red over the nodule.

## **Fate of chalazion:**

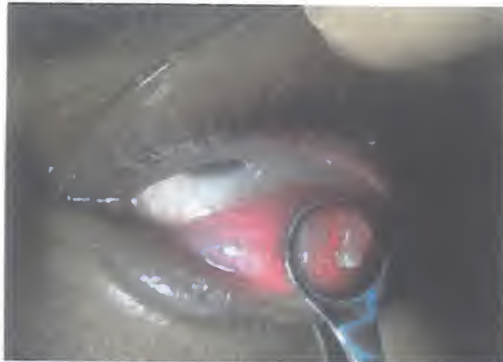
- 1) Spontaneous **resolution** is rare.
- 2) **Infection** forming an acute chalazion.
- 3) **Marginal chalazion:** The granulation tissue forms only in the duct and projects on the lid margin as a red nodule.
- 4) **Cyst formation.**
- 5) It may **open through the conjunctiva.**
- 6) It may reach a **large size** pressing on the globe and leading to astigmatism & mechanical ptosis.
- 7) **Multiple chalazia** may affect the whole tarsus.
- 8) **Malignant transformation.** (M.g. carcinoma).
- 9) **Mechanical ectropion** (if in lower lid).





### Treatment:

- 1) Very small chalazion: Vitamin A, local antibiotic and steroid preparation.
- 2) Marginal chalazion: Scraping from lid margin followed by diathermy.
- 3) Moderate or large chalazion: Vertical incision and scraping through the conjunctival side.
- 4) Multiple chalazia:  
Combined excision of tarsus and conjunctiva leaving the lower third of the tarsus (to avoid lid notching) with replacement by a mucous graft from the lip.
- 5) Recurrent chalazion of the same gland: Excision biopsy to exclude malignant tumor



## Disorders of eye lashes

- 1) Trichiasis: More than 4 lashes rubbing against cornea or conjunctiva.
- 2) Rubbing lashes: 4 lashes or less rubbing against cornea or conjunctiva.
- 3) Distichiasis: Extra row of lashes related to meibomian glands.
- 4) Poliosis: Whitening of the lashes.
- 5) Madarosis: Permanent absence of eyelashes due to destruction of lash follicle.

### Causes:

- 1) **Local:** inflammation (stye, trachoma), traumatic (burn), surgical (diathermy).
- 2) **General:** Alopecia, Myxedema, Syphilis and Leprosy.

Treatment: remove the cause and artificial lashes.



Distichiasis



Poliosis



Madarosis

# Trichiasis

## **Etiology:**

1) **Congenital trichiasis:** Often in all 4 lids. It is called Distichiasis.

In this condition, an extra row of lashes is present behind the gray line in the place of the ducts of the meibomian glands.

2) **Acquired trichiasis:** may be caused by the following:

- a) Trachoma (**commonest cause**) due to fibrosis distorting the hair follicles.
- b) Ulcerative blepharitis.
- c) Burns.

## **Clinical picture:**

### **Symptoms:**

- 1) Foreign body sensation.
- 2) Photophobia.
- 3) Lacrimation.
- 4) Blepharospasm

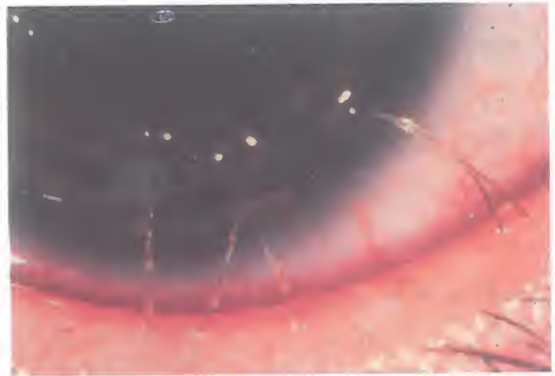
### **Complications:**

1) **Conjunctiva:**

- a) Chronic conjunctivitis.
- b) Conjunctival ulcer.
- c) Epithelial plaque.

2) **Corneal:**

- a) Recurrent ulceration leading to corneal opacities.
- b) Superficial vascularization.
- c) Epithelial plaque.



## **Treatment:**

1) **Rubbing lashes:** When 4 lashes or less are rubbing, treatment is to destroy the hair follicle by:

- a) Thermal coagulation by diathermy.
- b) Chemical coagulation by electrolysis due to release of NaOH.
- c) Cryo-coagulation.
- d) Epilation: pulling out of the lash is not a permanent treatment since the lash grows again in 4-6 weeks.

## Electrolysis

- ✚ The +ve electrode is a wet pad of cotton applied to any part of the body,
- ✚ The -ve electrode is a needle introduced into the hair follicle. The current is switched 2.5 mA for 10 seconds, coagulating the hair follicle and the lash can be easily pulled out.
- ✚ This occurs through chemical reaction as (- ve) electrode attracts (+ve) ions (Na) which reacts with  $H_2O$  to form NaOH which destroy hair follicle.

## Diathermy

- ✚ One electrode (a broad piece of lead) is tied to any part of the body.
- ✚ The second electrode is a needle, which is introduced into the hair follicle,
- ✚ The current is switched to 30 mA for 3 seconds.
- ✚ The follicle is coagulated and destroyed; the hair follicle and the lash can be pulled out easily.

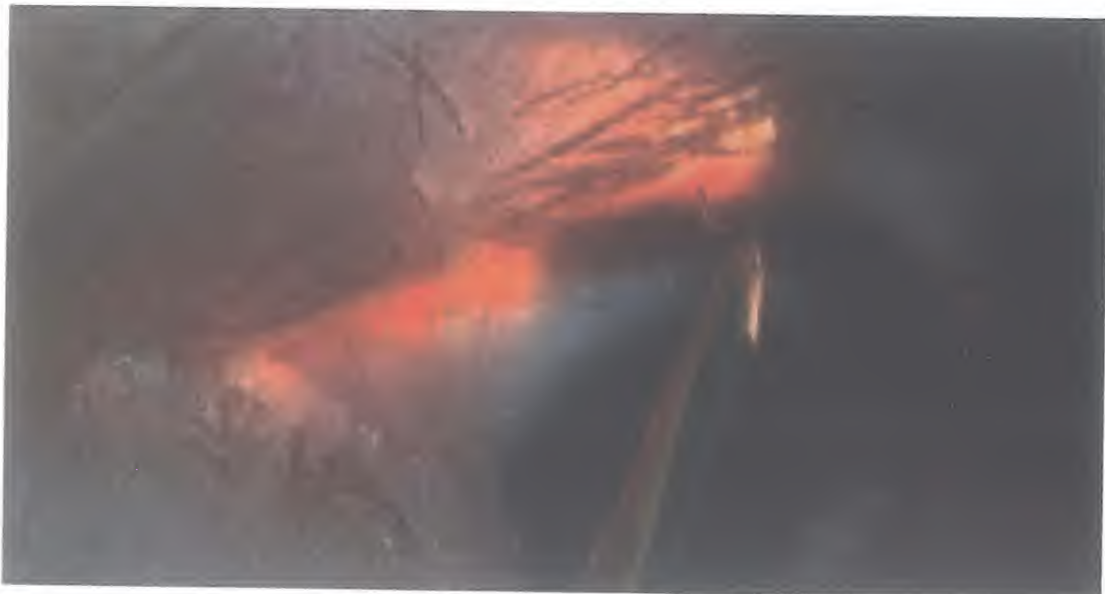
### 2) Trichiasis:

#### a) In the upper lid: (Van Millengen's operation):

**Principle:** Displace the rubbing lashes away from the cornea by placing a buccal mucous graft in the gray line.

#### b) In the lower lid: (Webster's operation):

**Principle:** Straighten the tarsus and lengthen the palpebral conjunctiva by placing a buccal mucous graft in an incision in the sulcus subtarsalis.





# Malpositions of the eye lid

## 1 - Entropion & Ectropion

	Entropion	Ectropion
<b>Definition</b>	Rolling inwards of the eyelid. The whole row of the lashes will be rubbing against cornea and sclera.	Rolling outwards of the eyelid from the globe. It usually affects the lower lid as it stands against gravity.
<b>Types</b>	<p><b>1) Cicatricial (fibrotic):</b> Fibrosis of the palpebral conjunctiva due to: Trachoma, chemical burns, diphtheria and ocular cicatricial pemphigoid</p> <p><b>2) Spastic:</b> Due to: two factors:  <b>a) Spasm of orbicularis muscle in response to ocular irritation</b> e.g. inflammation, exposed sutures, operations. It may be temporary or permanent.  <b>b) Lack of support of the eyelids by the globe following enucleation or enophthalmos.</b></p> <p><b>3) Involutional (Senile):</b> Affects only the lower lid due to overriding of the pre-septal portion of orbicularis muscle over the pretarsal portion.</p> <p><b>4) Congenital:</b> Usually affecting the whole lower eyelid (D.D. epiblepharon: extra horizontal fold of skin stretching along lid margin medially)</p>	<p><b>1) Cicatricial (fibrotic):</b> Due to: Scarring and contracture of the skin of the lower lid by burns, trauma or tumor</p> <p><b>2) Paralytic:</b> Due to paralysis of orbicularis muscle in facial nerve paralysis</p> <p><b>3) Involutional (Senile):</b> Due to senile weakness of the orbicularis muscle and relaxation of the palpebral ligaments.</p> <p><b>4) Congenital (rare)</b></p> <p><b>5) Mechanical:</b> Due to increased weight of lower lid e.g. multiple chalazia</p>



	Entropion	Ectropion
Clinical picture & Complications	<u>The same as in trichiasis</u>	<p><u>Symptoms:</u></p> <ol style="list-style-type: none"> <li>1) Bad cosmetic appearance</li> <li>2) Epiphora → eczema → ectropion → more epiphora and eczema</li> </ol> <p><u>Signs:</u> depending on the degree of ectropion:</p> <ol style="list-style-type: none"> <li>1) <u>Mild:</u> Exposure of the lower punctum.</li> <li>2) <u>Moderate:</u> Exposure of palpebral conjunctiva.</li> <li>3) <u>Severe:</u> Exposure of bulbar conjunctiva.</li> </ol> <p><u>Complications:</u></p> <ol style="list-style-type: none"> <li>1) Loss of marginal strip of tears and epiphora</li> <li>2) Chronic conjunctivitis and xerosis</li> <li>3) Ulceration and opacification of the lower part of cornea.</li> </ol>
Treatment	<p><u>1) Cicatricial:</u></p> <p>a) <u>In the upper lid:</u></p> <ul style="list-style-type: none"> <li>❖ <u>Snellen's operation:</u> Removal of a wedge of the tarsus. The edges are approximated by sutures, everting the lid.</li> <li>❖ <u>Webster's operation.</u></li> <li>❖ <u>Anterior lamellar reposition.</u></li> <li>❖ Transverse tarsotomy with marginal rotation (<u>Wise procedure</u>)</li> </ul> <p>b) <u>In the lower lid:</u></p> <ul style="list-style-type: none"> <li>❖ <u>Webster's operation.</u></li> <li>❖ <u>Wise procedure</u></li> </ul>	<p><u>1) Involutional (senile)</u></p> <p>a) <u>Mild cases:</u></p> <ul style="list-style-type: none"> <li>❖ <u>Instruct the patient to wipe his lower lid upwards</u></li> <li>❖ <u>Cautery punctures to the palpebral conjunctiva</u> to induce fibrosis and contraction of the conjunctiva with correction of ectropion.</li> <li>❖ <u>Snellen's inverting sutures:</u> to invert the lid</li> </ul>

## 2) Spastic:

a) Treatment of any cause of irritation e.g. ttt of corneal ulcer.

### b) Mild cases:

❖ T-shaped plaster: This method is ineffective as it becomes loose.

❖ Lateral canthotomy: to weaken Riolar's muscle temporarily.

### c) Recurrent cases:

❖ Lateral canthoplasty to weaken Riolar's muscle permanently.

❖ Skin and muscle operation: where an elliptical area of skin and orbicularis muscle of the lower lid is removed (This operation can be used in congenital entropion).

## 3) Involutional:

❖ Everting sutures: temporary treatment in mild cases.

❖ Jones procedure: tucking of lower lid retractors

❖ Lateral canthal sling: Fixation of lateral part of tarsus to lateral orbital rim

### b) Severe cases:

❖ Lateral canthal sling: very effective to support lower lid.

❖ Horizontal lid shortening and blepharoplasty.

## 2) Cicatricial (fibrotic):

a) Small scar: V to Y Plasty or Z plasty.

b) Large scar: Skin graft (donor sites: post auricular area or upper inner arm)

## 3) Paralytic (facial palsy):

a) Protection of the cornea: by drops during the day and ointment during sleep.

### b) Medical treatment:

❖ To help nerve regeneration by cortisone.

❖ Anti-rheumatic medication and massage.

c) Lateral tarsorrhaphy: to induce adhesion between upper and lower lids at the lateral canthus either temporary or permanent to narrow the palpebral fissure.

### d) Fascia lata sling and silicone sling operation:

in severe and recurrent cases, where a sling is passed between the medial and lateral palpebral ligaments to support the lower lid in its normal position.

4) Mechanical: Treat the cause





**Congenital Entropion**



**Congenital Ectropion**



**Cicatricial Entropion**



**Cicatricial Ectropion**



**Senile Entropion**



**Senile Ectropion**



**Paralytic Ectropion**



**Mechanical Ectropion**



# PTOSIS

**Definition:** Dropping of the upper eye lid that normally covers the upper  $\frac{1}{6}$  of the cornea

## **Etiology:**

### **1) Myogenic:**

a) **Congenital ptosis** due to dystrophy of levator muscle leading to poor contraction and incomplete relaxation



b) **Acquired ptosis: Myasthenia gravis:** due to a defect in the myoneural junction. It is bilateral, increases at the end of the day and on prolonged fixation

### **2) Neurogenic:** due to a disorder of nerve supply

a) **3<sup>rd</sup> nerve palsy:** Paralytic ptosis → (Diabetes – congenital – Traumatic).

b) **Horner's syndrome:** due to disorder of sympathetic nerve supply.



### **3) Aponeurotic Ptosis:** Disorder in levator aponeurosis.

a) **Senile (Involutional):** degenerative process with age

b) **Postoperative ptosis:** Following cataract Surgery and retinal detachment surgery. (Damage of levator, superior rectus complex).

### **4) Mechanical Ptosis:**

a) **Excess weight** (edema - tumor- chalazion).

b) **Conjunctival scarring**



**Bilateral Ptosis**

## **Clinical Picture:**

**Symptoms:** Drooping of upper lid.

## **Signs:**

### **1) General appearance:-**

- a) Corrugations of the forehead.
- b) Arching of eyebrow. →
- c) Chin elevation.
- d) Face turn and head tilt



### **2) Assessment of ptosis:**

**Degree of ptosis: (Margin - reflex - distance): normally (4 - 4.5 mm)**

Mild	2 mm Dropping
Moderate	3 mm Dropping
Severe	4 - 4.5 mm Dropping

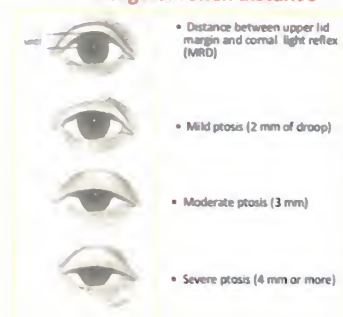
## Levator function: (excursion) test:

### 1 - Distance of U.L.: Movement from down gaze to up gaze. (Upper lid excursion):

15 mm	Normal	5 – 11 mm	Fair
12- 15 mm	Good	5 mm	Poor

### 2- Upper lid crease presence.

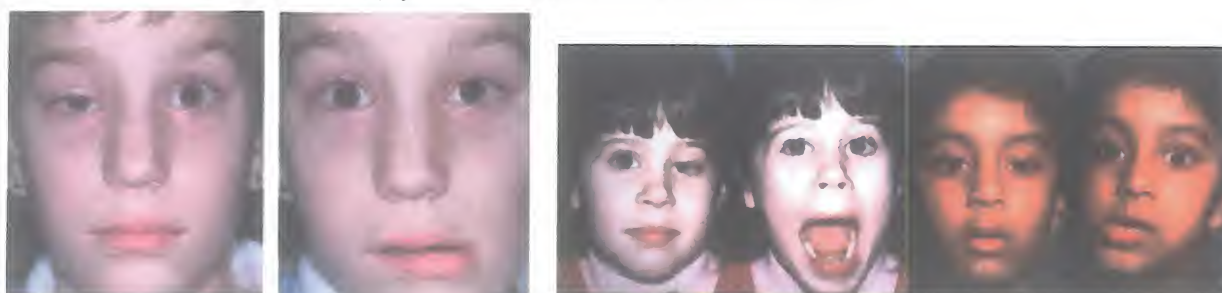
#### Marginal reflex distance



### 3) Associated signs:

#### a) Marcus – Gunn (jaw – winking) phenomenon:

The ptosed lid elevates on moving the jaw due to faulty innervation so that nerve fibers to pterygoid muscle supply levator muscle instead of 3<sup>rd</sup> nerve.



#### b) Ocular Motility: Limitation of ocular motility upwards due to weak or paralyzed superior rectus. Limitation of ocular motility may be also due to 3<sup>rd</sup> nerve palsy.

#### N.B.1: Superior rectus & levator

1. Arises from the same dermatome.
2. Included in the same sheath.
3. Nerve supply to levator passes through SR.

#### N.B.2: 3<sup>rd</sup> nerve supplies [Levator muscle- MR- IR- SR]

#### c) Bells phenomenon.

#### d) Corneal sensation.

#### e) Squint.

#### f)

#### g) Increased innervation.

#### h) Pupil: Ptosis & Mydriasis → 3<sup>rd</sup> nerve palsy.

Ptosis & Miosis → Horner's syndrome.

### **Complications:**

- 1) Amblyopia (unilateral congenital ptosis).
- 2) Lumbar lordosis.
- 3) Torticollis and contracture of sternomastoid.
- 4) Nystagmus & mental retardation (Bilateral congenital).

### **D.D: Causes of pseudo-ptosis:**

In contralateral side	In ipsilateral side
❖ Lid retraction	❖ Lack of support by globe (enophthalmos - Atrophia bulbi)
❖ Severe proptosis	❖ Hypotropia
	❖ Excessive dermatochalasis

### **Treatment:**

#### **1) Congenital ptosis: (factors affecting the line of treatment and prognosis)**

- a) Squint if present → squint surgery first to avoid diplopia.
- b) Hypotropia if present → correct hypotropia.
- c) Unilateral severe ptosis → Do ptosis surgery as early as possible (to avoid amblyopia).
- d) Jaw winking phenomenon → Do frontalis sling (never levator resection Surgeries

### **Surgeries**

#### **✚ Levator tucking:**

For mild ptosis with good levator function (levator function more than 10 mm).

- ✓ **Idea:** part of levator muscle is excised together with the upper border of tarsus through conjunctiva.

#### **✚ Levator resection:**

1. Can be made from skin side (**Ever Bauch's**)
2. Can be made from conjunctival side (**Blascovics**).  
(Levator function from 5 mm to 10 mm).

- ✚ **Hess operation or frontalis sling:** Suspension of the upper lid on occipito frontalis muscle using endogenous material as fascia lata or exogenous material as 5.0 prolene sutures ( It is better used in bilateral cases)

#### **2) Acquired ptosis:**

- a) Mechanical → Remove the cause.
- b) Hysterical → Psychotherapy
- c) Myasthenia gravis → Prostigmine
- d) Paralytic ptosis → wait for 6 months + Vit. B complex therapy then evaluate ptosis and surgery if needed.



# Lagophthalmus

**Definition:** Incomplete closure of the lids.

## **Etiology:**

1) **Physiological** (lack of orbicularis tone during sleep).

2) **Pathological**

### a) Local causes:

- ✚ Paralysis of Orbicularis Oculi: (Facial palsy) or (Bell's palsy).
- ✚ Severe Ectropion.
- ✚ Scarring of the lid.
- ✚ Coloboma of the lid.
- ✚ Severe proptosis.



### b) General causes:

Severe weakness or illness or coma which leads to generalized atonia.

## **Clinical picture:**

1- Mild cases: Lagophthalmos can be overcome by forcible lid closure,

2- Severe cases: The eye is permanently opened which leads to:

- a) Chronic conjunctivitis and xerosis.
- b) Exposure keratopathy (exposure of the cornea during sleep → for dryness of its lower third)
- c) Corneal ulceration (oval horizontal ulcer involving lower third).

N.B.: Upper part of cornea is protected during sleep due to Bell- phenomenon

(Rolling up of the eye ball involuntary during sleep or forcible closure of the lid)

## **Treatment:**

- 1) **Treatment of the causes.**
- 2) Protection of the cornea against dryness during day by glasses or C.L and during sleep by eye ointment.
- 3) **Tarsorrhaphy:** suturing lid margins together, this may be temporary or permanent.



# Symblepharon

**Definition:** Adhesions between the lid and the globe, or adhesions between bulbar or palpebral conjunctiva and the cornea

**Etiology:** It is due to the presence of two raw surfaces as in:

- 1) Burns and caustics.
- 2) Post inflammatory e.g. trachoma and diphtheria.
- 3) Post operative e.g. Pterygium operation.
- 4) Ocular cicatricial pemphigoid.

**Types of symblepharon:**

- 1) Anterior symblepharon → Adhesion between lid & cornea or bulbar conj.
- 2) Posterior symblepharon → Adhesion at the fornix "trachoma".
- 3) Total symblepharon → the whole palpebral with bulbar conj.

**Clinical picture:** 3 D

- 1) Disfigurement.
- 2) Diminution of vision.
- 3) Diplopia due to limitation of ocular motility.

**Treatment:**

1) **Prophylactic treatment:**

- a) **Ointment:** put ample amounts of ointment during day and night.
- b) **Corticosteroids:** anti fibroblastic (in absence of infection).
- c) **Glass rod coated with antibiotic ointment** is passed between the lid and the globe several times daily.
- d) **Contact shell** is used until healing occurs.

2) **Actual treatment:**

- a) **Synechotomy:** cut the adhesions.
- b) **Mucous membrane graft:** to Cover the 2 opposite surfaces.
- c) **Keratoplasty:** if the cornea is opaque.



## Xanthelasma

**Definition:** Subcutaneous deposits of cholesterol in the medial canthus.

**Incidence:** Seen in diabetics and patients with hypercholesterolemia.



## Dermatochalasis

**Definition:** Redundancy of upper eyelid skin in old age.

**Treatment:** Blepharoplasty.



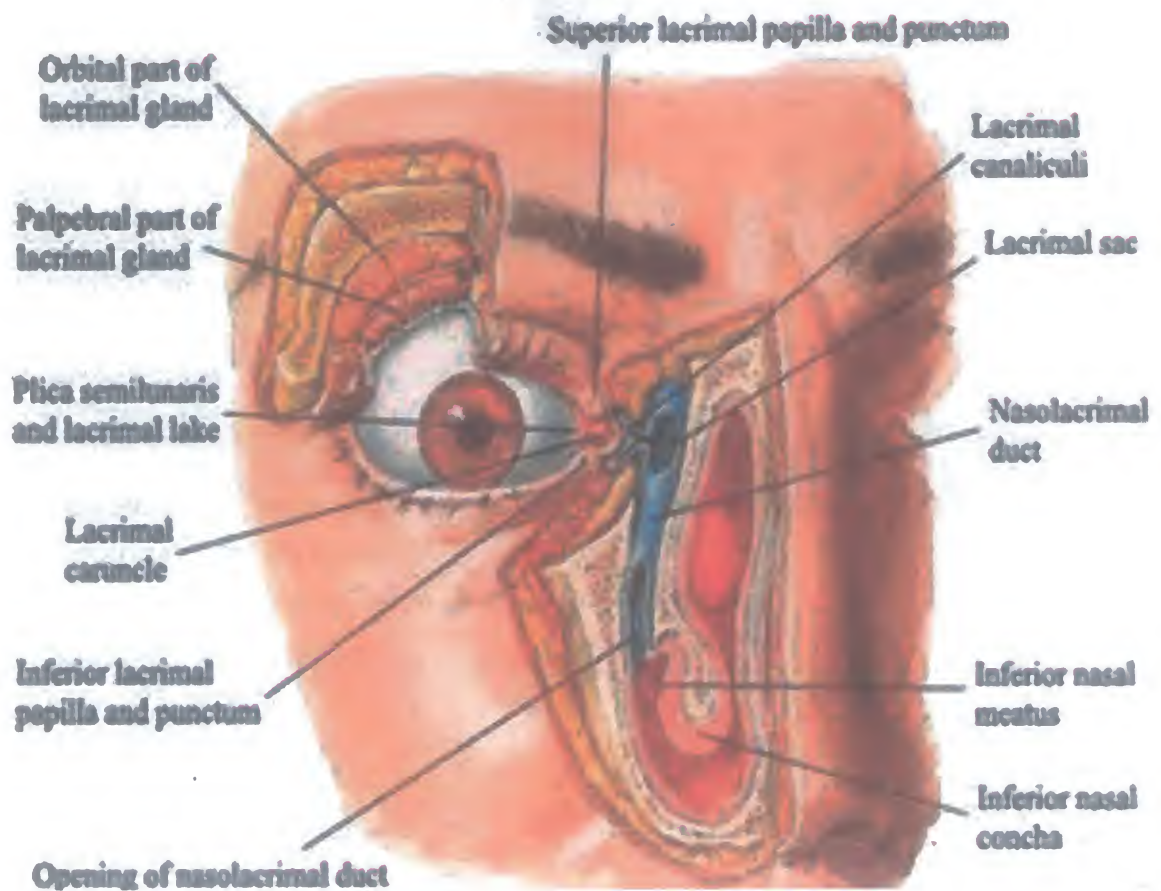
## Blepharochalasis

Recurrent attacks of upper lid edema, leading to redundancy of upper lid skin in young age



# Lacrima!

## Lacrimal apparatus Dissection



# The Lacrimal apparatus

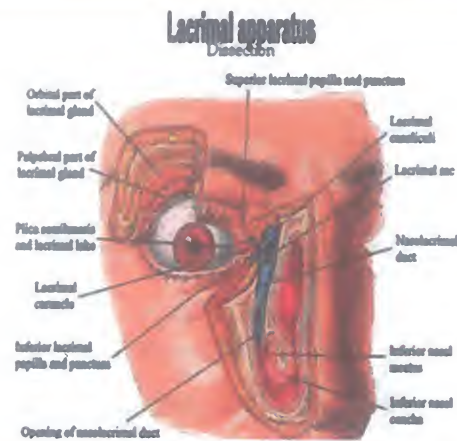
The Lacrimal apparatus consists of:

## 1- Secretory system:

- a) Lacrimal gland.
- b) Goblet cells.
- c) Accessory lacrimal glands.

## 2- Drainage system:

- a) 2 puncti.
- b) Lacrimal Sac.
- c) 2 canaliculi.
- d) Naso-lacrimal duct



## 1 - The Lacrimal Secretory System

### 1-The main lacrimal gland:

#### a) Gross anatomy

- ✚ **Site:** In a shallow bony fossa in the antero-lateral part of the orbital roof.
- ✚ **Shape:** Almond shaped.
- ✚ **Parts:** The gland is divided by the levator aponeurosis into 2 parts:
- ✚ **Orbital portion:** Superior & larger.
- ✚ **Palpebral portion:** Inferior & smaller (rests on sup. fornix & can be seen if the upper lid is everted).
- ✚ **Ducts:** To 10-12 ducts arise from orbital portion & pass through palpebral portion to open in the lateral part of the upper Fornix.  
(So removal of palp. portion prevents secretion of whole gland).

#### b) Microscopic anatomy The gland is formed of acini which are lined by 2 layers of cells:

- ✚ **Outer flat myo-epithelial cells** (contractile).
- ✚ **Inner columnar** (Secretory).

#### c) Blood supply Lacrimal artery (branch from the ophthalmic artery).

#### d) Nerve supply

- ✚ **Sensory:** Lacrimal nerve (branch from ophthalmic n.).
- ✚ **Parasympathetic:** Secretory.
- ✚ **Sympathetic:** (Vasomotor): from the superior cervical ganglion.

#### e) Lymph Drainage Pre-auricular lymph nodes.

#### f) Function Responsible for 95% of tear secretion.

## 2- Accessory lacrimal glands:

These are microscopic glands.

### Glands of Krause:

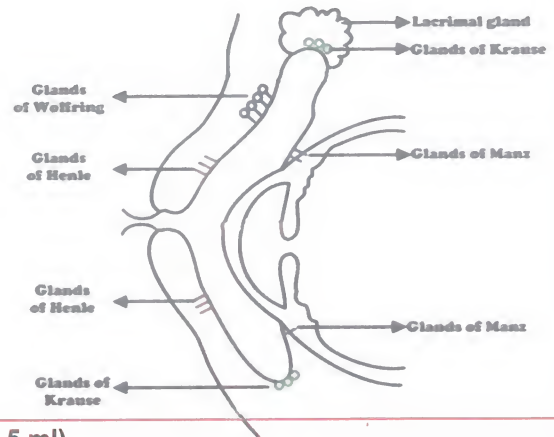
Site: Upper and lower fornices.

Number: 40 (Upper F.) & 10 (Lower F).

### Glands of Wolf ring:

Site: Upper border of upper tarsus.

Number: 5



N.B.1: Amount of tear secreted per day is about: (1 - 1.5 ml).

N.B.2: PH of tears is slightly alkaline.

N.B.3.: The pre-corneal tear film made of 3 layers:

- 1) Oily layer: From meibomian gland (see lid).
- 2) Watery layer: From lac. gland (lubrication, nutrition, protection & optical).
- 3) Mucin layer: From goblet cells (change corn, epith. From Hydrophobic to hydrophilic).

## 2- The Lacrimal drainage system

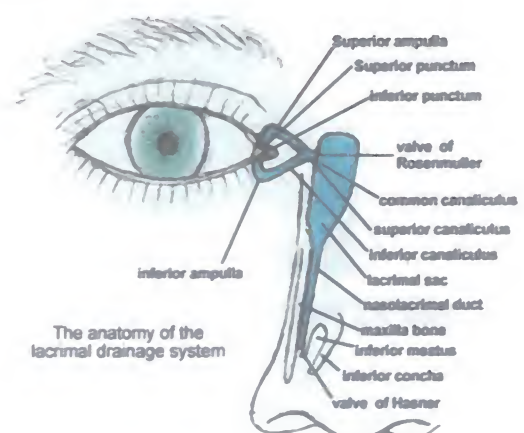
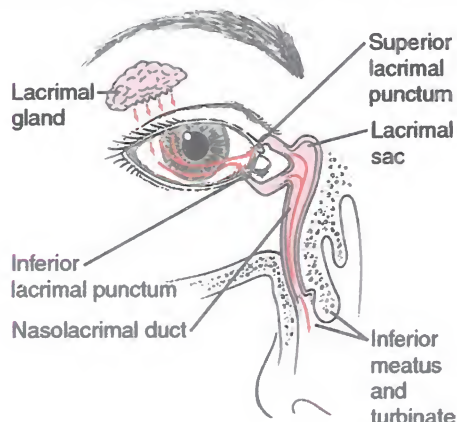
### 1- Gross anatomy:

#### a) Puncti:

- ✚ Present on lid margin near its posterior border.
- ✚ At a distance of 6 mm lateral to medial canthus on 2 elevations called lacrimal papillae.
- ✚ Not visible (normally) except if lids are pulled away from eye or in ectropion.

#### b) Two canaliculi:

- ✚ These are fine tubes which carry tears from the puncti to the lac. sac.
- ✚ Each canaliculus is formed of 2 portions
  - ✓ Vertical: 2 mm.
  - ✓ Horizontal:
- ✚ The 2 canaliculi open into the lacrimal sac by a common canaliculus which opens at the junction between the upper  $\frac{1}{3}$  and the lower  $\frac{2}{3}$  of the sac.





## c) Lacrimal sac

✚ **Site:** In lac. fossa (in medial orbital wall).

✚ **Size:** When distended its (8 x 12 mm).

✚ **Parts:**

i. **Fundus:** Upper part (extends above med palp, ligament).

ii. **Body:** Main part.

iii. **Neck:** Narrow & continuous with NLD. It is enclosed in the lacrimal fascia.

iv. **Naso lacrimal duct (NLD):**

✓ It is (12 - 24 mm) tube which carries tears from lac. sac to the nose.

It opens in inferior meatus.

✓ Direction: downward, backward & laterally.

✓ Its opening in the nose is guarded by a valve (Hasner's valve).

## 2- Microscopic anatomy:

a) **Puncti and Canaliculi:** Are lined by St. Squamous epith.

b) **Sac and Nasolacrimal duct:** Are lined by 2 layers of col. epithelium contains goblet cells.

c) **N.L. duct:** Is surrounded by C.T. rich in veins. Congestion of this plexus lead to NLDO

## Blood supply:

Arteries: Supratrochlear, Infraorbital and External nasal arteries,

Veins: Infraorbital, Angular & Nasal veins.

Nerve supply: 5<sup>th</sup> nerve

Lymphatic drainage: To Submaxillary lymph nodes

## Tear drainage:

1) Evaporation: 25% of tears.

2) Excretion:

a) **Passive:** gravity & capillarity.

b) **Active:** Lacrimal pump through orbicularis ms

# Diseases of lacrimal passages

## Watering of the eye

**Definition:** It means over-flow of tears over the cheek due to either:

Increased secretion (Lacrimation)

OR

Decreased drainage (epiphora)

## Lacrimation

1- **Emotional:** Excitement.

2- **Reflex:** Due to corneal ulcer – iritis – corneal or conjunctival F.B.

## Epiphora

**Etiology:**

### 1) Diseases of the upper lacrimal passages:

#### a) Lids:

- ❖ Ectropion
- ❖ Irregular posterior border
- ❖ Orbicularis Paralysis (pump failure)

#### b) Punctum: Stenosis or obstruction either:

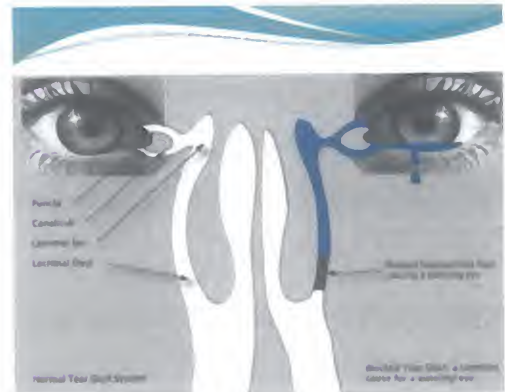
- ❖ Congenital
- ❖ **Acquired:** infection as in Trachoma.

#### c) Canaliculus: Stenosis or obstruction either:

- ❖ Congenital.
- ❖ Post inflammatory and post traumatic occlusion.
- ❖ Foreign body: Lash.

#### ❖ Fungal canaliculus:

- ✓ Caused by Actinomyces.
- ✓ The punctum is red and prominent.
- ✓ Gritty sensation on probing
- ✓ On pressure: A mucopurulent discharge with black concentration or sulfur granules may be expressed.



### 2) Diseases of the lower lacrimal passages:

#### a) Lacrimal Sac:

- ❖ Congenital absence
- ❖ Adhesion from repeated inflammation.
- ❖ Tumors of the lacrimal sac.
- ❖ Fracture of lacrimal bones.

### b) Naso-lacrimal duct:

- ❖ **Congenital:** imperforate Hasner's valve.
- ❖ **Acquired:**
  - ✓ Venous congestion (50 % of cases of epiphora)
  - ✓ Adhesions.
  - ✓ Tumors of bony canal.

### c) Nose:

- ❖ **Polyps or tumors.**
- ❖ **Deviated septum.**

## **Investigations:**

### 1) Ocular examination:

- a) **Exclude causes of lacrimation.**
- b) **Examine lid margin** (for ectropion, irregularities, punctual obst.).

### 2) Diagnostic tests:

#### a) Regurge test:

- ❖ Ask patient to look up (to evert punctum).
- ❖ Press by a little finger on the lacrimal sac below the medial palpebral ligament: (backward, medially & upward)
- ❖ If:
  - False (-ve) reg. → **upper segment obstruction.**
  - (+ve) reg. → **NLD obstruction.**

#### b) Fluorescein test:

- ❖ Put a drop of fluorescein in the conj. sac & after 2 minutes.
- ❖ Ask patient to blow his nose in a white handkerchief.
  - Green color is seen: **patent passages.**
  - No Green color is seen: **obstruction** (but the level is not determined).



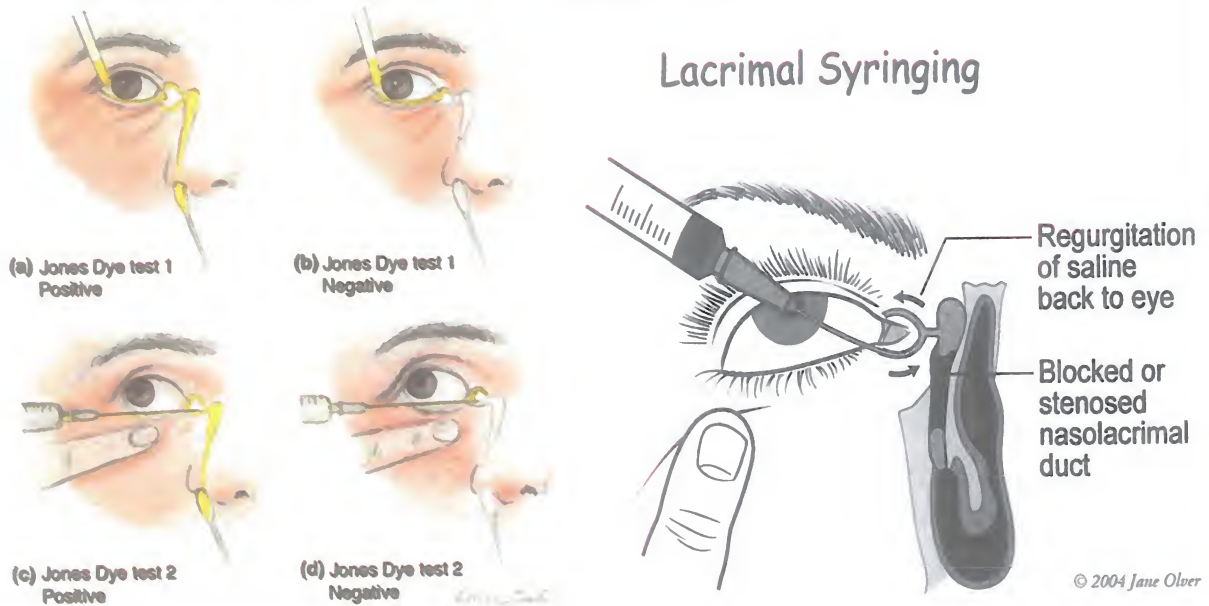
#### c) Diagnostic syringing:

- ❖ Dilatation of lower canaliculus, then syringing with saline is done:
  - Regurge from upper punctum → **NLD obstruction.**
  - Regurge from same punctum → **canalicular obstruction.**



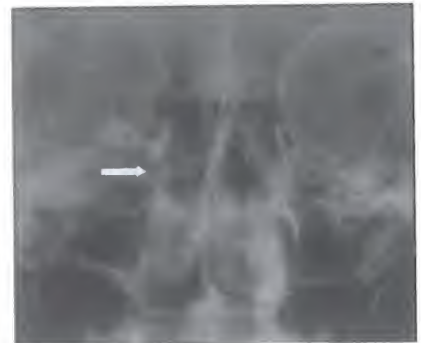
#### d) Jones-dye test:

- ❖ **Test (1):** Fluorescein test is done if no green color.
- ❖ **Test (2):** The lacrimal passages are irrigated with saline.
- ❖ **If:**
  - Fluorescein is recovered → **partial obstruction or pump failure.**
  - No saline reach nose → **complete obstruction.**



#### e) Dacryocystography:

- ❖ A radio-opaque dye (lipidol) is injected in the canaliculus and repeated
- ❖ X-rays are taken.
- ❖ **If:**
  - Level of the dye is seen → **obstruction.**
  - Rat tail appearance → **stenosis.**
  - Delayed emptying → **failure of lacrimal pump.**
- ❖ **Normal emptying:** (Sac: 15 min) - (Duct: 30 min).



#### f) X-ray on the nose & sinuses: for ENT examination

#### Treatment:

1) Lid & nose causes: Treatment of the cause.

2) Punctum causes:

a) **Stenosis:**

- ❖ Dilatation is done by lacrimal dilator.
- ❖ On snip ampullotomy

b) **Obstruction:**

❖ Do the 3 snip operation.

❖ Laser punctoplasty

### 3) Canaliculus causes:

a) **Stenosis:** dilatation is done by probing.

b) **Obstruction:** do conjunctivo-DCR.

### 4) Naso-lacrimal duct obstruction:

#### a) Congenital:

✚ **First 6 m.:** Antibiotic drops + massage (toward nose hoping for spontaneous cure).

✚ **6 - 12 m:** probing + syringing.

✚ **DCR:** if repeated probing failed (dacryo - cysto - rhinostomy).

#### b) Acquired:

✚ **TTT of the cause.**

✚ **Probing.**

✚ **DCR.**

### Probing

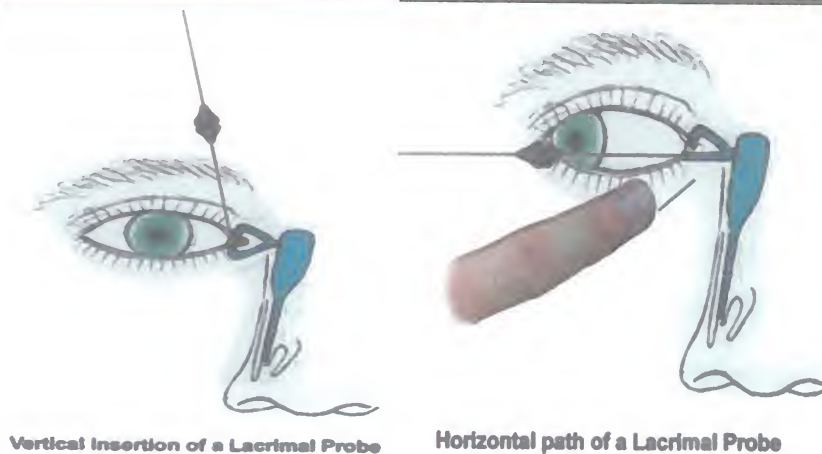
1) **Anesthesia:** General (young), local (adult).

2) **Dilate the punctum by lacrimal dilator.**

3) **Introduce the probe in the anatomical direction of lacrimal passages:**

✚ **First:** Vertically (2mm) then horizontally until it hits the bone (of lacrimal fossa).

✚ **Then:** Withdraw slightly & direct it down & slightly lateral & backward.



# Inflammation of the lacrimal sac

## Acute dacryocystitis

**Definition:** It is acute suppurative inflammation of the sac.

**Etiology:** Organism: usually **Staph.** (from the conjunctiva) – may be **pneumococci** or **streptococci** (Either: De novo or on top chronic inflammation).

**Clinical picture:**

### Symptoms:

1) **General:** Fever & Headache.

2) **Local:**

Painful swelling at the site of the sac & epiphora.

### Signs:

1) **Swelling:**

Red, Hot & tender below the medial canthus.

2) **Regurge test:** -ve (due to edema of canaliculi)



### **Complications:**

- 1) Lacrimal fistula.
- 2) Orbital cellulitis.
- 3) Cavernous sinus thrombosis.
- 4) Become chronic.

### **Treatment:**

**General:** Antibiotics.

### **Local:**

- 1) Hot fomentation.
- 2) Pus: Drain (skin incision over the sac).
- 3) Fistula: Fistulectomy + Dacryocystorhinostomy





# Chronic dacryocystitis

**Definition:** It is chronic inflammation of the lacrimal sac.

## **Etiology:**

- 1) **Stasis of tears in the sac:** due to NLD obstruction (Cong, or acquired).
- 2) **Infection:**
  - a) pneumococci (80% from nose, sinuses).
  - b) Staph. – Strept. – Trachoma – Fungi.
  - c) TB and syphilis

**N.B. Chronic dacryocystitis is more in menopausal females, this is because:**

- 1) NLD is anatomically narrower.
- 2) Endocrinal disturbances at menopause leading to thickening of the lining mucosa.

## **Clinical picture:**

**Symptoms:** Fullness + watery eye

### **Signs:**

- 1) Swelling (below the medial canthus).
- 2) Regurge test: (+ve)  
→ First watery (tears), then mucopurulent, lastly frank pus.



## **Complications:**

- 1) **Lid:** Epiphora (vicious circle).
- 2) **Conj.:** Chronic conjunctivitis.
- 3) **Cornea:** Ulcer (Hypopyon).
- 4) **Lacrimal sac:**
  - a) Acute dacryocystitis.
  - b) Obstruction of canaliculi Mucocoele.



Mucocoele is a collection of mucous from dying goblet cells

- 5) **Endophthalmitis:** If not treated before intra-ocular operations.

## **D.D.:**

- 1) Cong, dacryocystitis from ophth. Neonatorum: by regurge test.
- 2) Mucocoele from dermoid cyst: by the site of the swelling.  
(Swelling below med. palpebral ligament → mucocoele).

## **Treatment:**

**Aim:** Is to restore communication between the sac & the nose as the obstruction prevents subsidence of the inflammation.

**By:**

### **1) Medical treatment:**

- a) Antibiotics.
- b) Vasoconstrictor nasal drops.

### **2) Surgical treatment:** Treatment of NLD obstruction: see epiphora.

## Operations

### **1) Dacryocystorhinostomy (DCR):**

**Principle:** Is to make anastomosis between the sac & middle meatus of the nose.

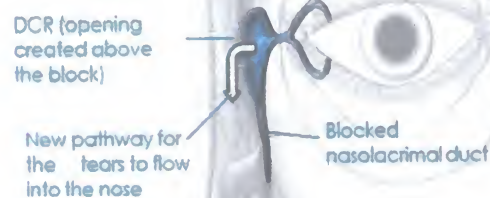
#### **Indications:**

- 1) Chronic dacryocystitis with relatively healthy sac & nose.
- 2) NLD obstruction (conj. or acquired) if probing is failed.
- 3) Mucocele.

#### **Contraindications:**

- 1) Bad condition of the sac (adhesions) & the nose (atrophic rhinitis).
- 2) Hypopyon ulcer.
- 3) TB or Tumors of the sac.

Dacryocystorhinostomy (DCR)



## Lacrimal intubation procedure

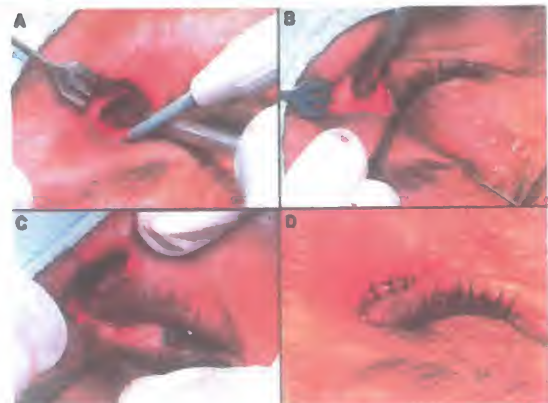
**Aim:** is permanent fistula between lac. Passages & the nose.

**By:** Silicone tube (removed after 3-6 months)

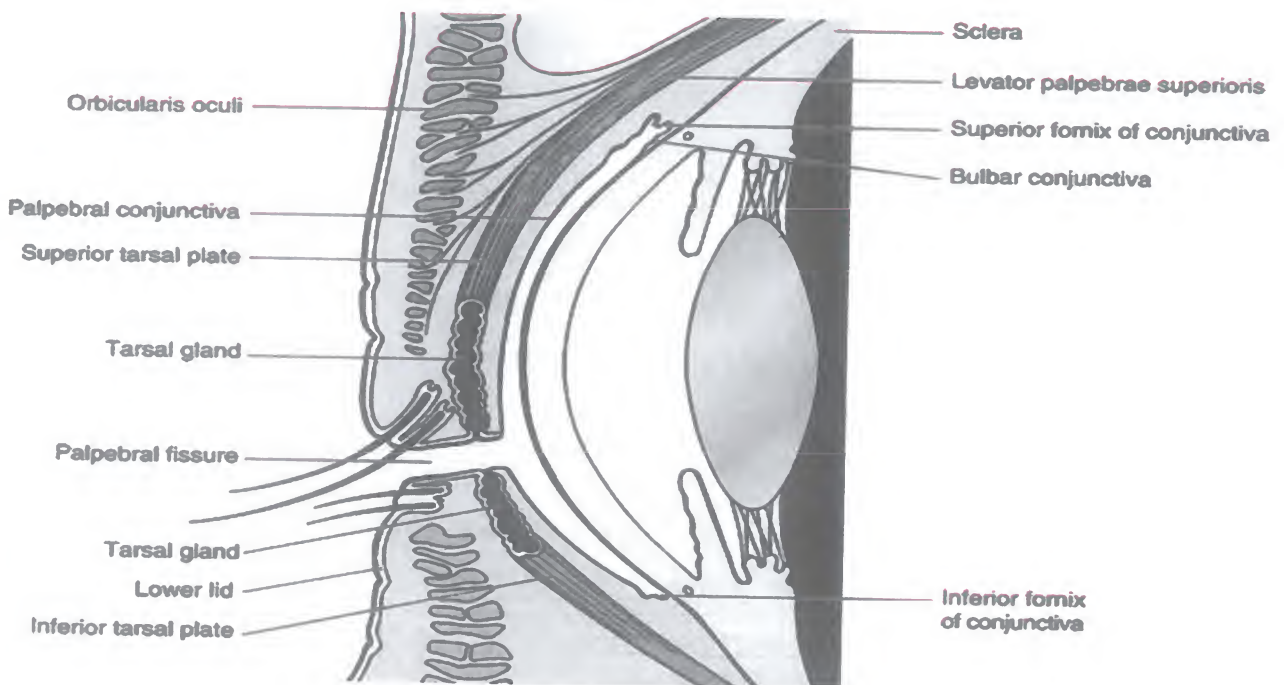
### **2) Dacrycystectomy:**

**Principle:** the sac is excised.

**Indications:** Are contra-indications of DCR.



# Conjunctiva





# Conjunctiva

## Anatomy

✚ The conjunctiva is a thin semi transparent mucous membrane lining the inner surface of the eye lids and reflected at the fornix to cover the anterior part of sclera. When the lids are closed, it forms the conjunctival sac

**Gross anatomy:** Conjunctiva is divided into the following parts:

- 1) **Palpebral conj.:** It lines the posterior surface of the lid and is formed of:
  - a) **Marginal part:** Starts at the gray line of the lid margin and continues the sulcus subtarsalis.
  - b) **Tarsal part:** Which is very vascular and adherent to the tarsus.
- 2) **The conjunctival fornix:** It is the reflected part bet. the lids and the globe and formed of: (Superior, Inferior, Medial and Lateral fornices).
  - ❖ **Superior fornix:** Is the deepest one and receives the openings of lacrimal gland ducts and insertion of L.P.S.muscle.
- 3) **Bulbar conjunctiva:** The bulbar conjunctiva covers the anterior part of the sclera and ends at the limbus, It is loosely adherent to sclera except 4 mm around the limbus are firmly adherent.
- 4) **Plica Semilunaris:** It is a delicate vertical crescent-shaped fold at the medial canthus corresponding to a rudimentary third lid seen in many animals.
- 5) **Caruncle:** Is a small wart-like, red structure present at the inner canthus, with certain skin characteristics including fine hairs and sebaceous glands.

**Minute anatomy:** The conj. is made of 2 layers:

- 1) **Epithelium:**
  - a) From lid margin to the sulcus subtarsalis (Marginal part) **stratified squamous ep**
  - b) From sulcus subtarsalis to fornix **2 layers of epithelium.**
  - c) From fornix to limbus **gradual increase in number of layers till the 3 mm** around limbus, where the epith. of conj. is similar and continuous with that of the cornea. **(5 – 6 layers of non keratinized stratified squamous epith.).**

**N.B. Between Epith. Cells of conjunctiva there is unicellular mucous glands (Goblet cells)**

**which secrete mucin layer of the tear film**

- 2) **Substantia propria:** Two layers:
  - a) **Superficial adenoid layer:** formed of loose fine C.T. rich in lymphocytes and develops three months after birth.
  - b) **Deep dens fibrous layer:** housing the nerves & vessels of conj.

## **Blood supply & lymphatic drainage:**

### **1) The conjunctiva receives its arterial supply from:**

- a) **Posterior conjunctival arteries:** which arise from the palpebral arteries to supply most of the conjunctiva except near the limbus.
- b) **Anterior conjunctival arteries:** which arise from the anterior ciliary arteries and supply the conjunctiva around the limbus.

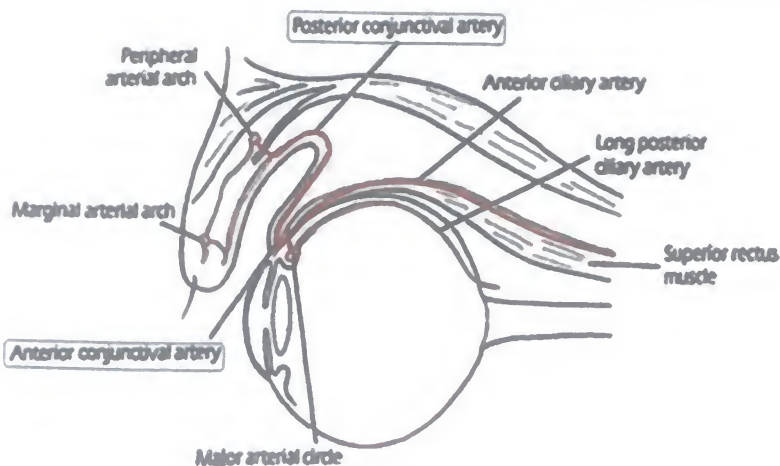
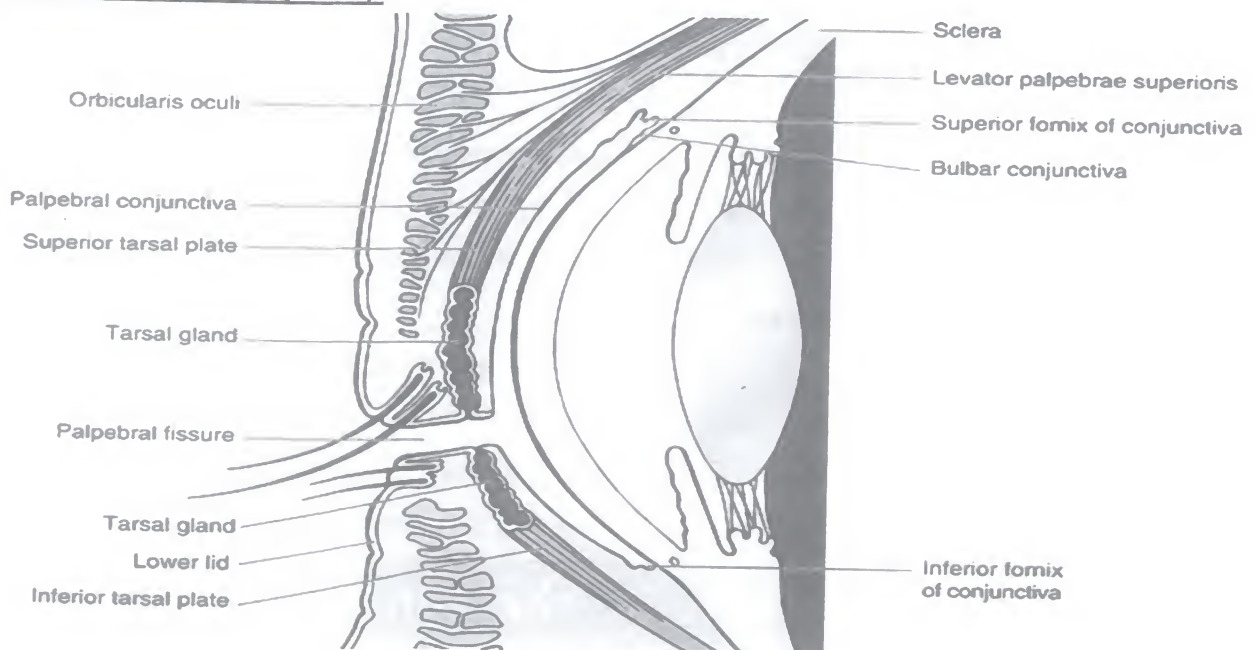
### **2) Venous drainage:** To the palpebral and ophthalmic veins.

### **3) Lymphatic drainage:** Parallels that of the lid:

- a) The lateral 2/3 drains into **the pre-auricular lymph nodes.**
- b) The medial 1/3, the caruncle and plica semilunaris drain into **the submandibular L.N.**

**Nerve supply:** The conjunctiva is supplied by **branches of the trigeminal nerve.**

**(Similar to that of eyelids).**



# Conjunctivitis

✚ Inflammation of conj. characterized by conj. Hyperemia & Discharge

## Common manifestations of conj. Inflammation:

- 1) **Irritation:** Discomfort - Burning sensations - F.B. Sensations - Itching.
- 2) **Discharge:** Watery - Muroid - Mucopurulent - Purulent - Bloody purulent.
- 3) **Chemosis** (Conj. Edema).
- 4) **Conj. Congestion or Hyperemia:** Dilatation and congestion of posterior Conj. Vessels (therefore congestion is more marked at fornix and decreases gradually towards limbus).
- 5) **Follicles:** (see later).
- 6) **Papillae:** Non-specific response to chronic irritation leading to epithelial proliferation with a vascular core.
  - ❖ They are bumps each with a central core of vessels.
  - Caused by: chronic infections and atopy.
- 7) **Giant papillae:** (see later).
- 8) **Membranes:** Damage of the conjunctival vessels and epithelial surface by severe inflammation can cause exudation of fibrin, inflammatory cells, necrotic cells and serous fluid, forming a dirty grayish membrane on the surface. Removal of a membrane produces a raw bleeding surface. Causes are
  - a) Diphtheritic conjunctivitis.
  - b) Severe viral infections.
  - c) Alkali or acid burns.
  - d) Stevens-Johnson syndrome
- 9) **Pseudo membranes:** Formed by condensation of discharge and exudates over the conjunctival surface. They are easily removed leaving an intact epithelial surface.

## Inflammations of the conjunctiva (Conjunctivitis)

### Classification:

#### 1) Infective Conjunctivitis:

- a) **Acute:** Serous, Catarrhal, Mucopurulent, Purulent, Viral and Membranous.
- b) **Sub-acute:** Catarrhal, Angular and Follicular.
- c) **Chronic:** Follicular and Granulomatous.

#### 2) Non-infective Conjunctivitis:

- a) **Acute:** Mechanical injury, Allergic, Photophthalmia and Burns.
- b) **Chronic:** Simple catarrhal, Keratoconjunctivitis sicca, Vernal, Phlyctenular and Ocular pemphigoid.



# Acute infective conjunctivitis

## 1 - Mucopurulent conjunctivitis

### Etiology:

#### 1) Haemophilus egypticus (Koch. Week's bacillus):

Cause epidemics in Egypt in the following months → April, May, September and October.  
(Related to the fly breeding season).

#### 2) Staph, Strept. and pneumococci:

	Koch-week's	Pneumococci
Incidence	Epidemics in Egypt	Epidemics in Europe
Season	Summer	Winter
Sensitive to	Sulpha (not penicillin)	Sulpha & Penicillin

### Clinical picture:

#### Symptoms:

- 1) Conjunctival irritation: (Discomfort, F.B. Sensation).
- 2) Red eye.
- 3) Discharge.

#### Signs:

- 1) Eyelids: (edema, lashes glued together in a pencil like appearance).
- 2) Conj.:
  - ❖ Edema (chemosis)
  - ❖ Congestion (hyperemia)
  - ❖ M.P. discharge.
- 3) General signs:
  - ❖ Pre-auricular lymphadenopathy.
  - ❖ Petechial hemorrhage

### Fate & Complications:

- 1) Spontaneous cure within 2 wks.
- 2) Marginal corneal ulcer (?? Why).
- 3) Chronicity.



## Management:

### 1- Prophylaxis:

- Combat flies.
- Protect the fellow eye by **putting E.D. in both eyes** starting by the normal one.
- Usage of **special utensils or towels**.

### 2- Treatment:

- Boric acid lotion 4% OR Sterile water** to remove discharge.
- Topical antibiotic E.D.:-**
  - ❖ Sulpha cetamide E.D. for *H. egypticus*.
  - ❖ Sulpha or broad spectrum for other.
- Ab E.oint. by night:**
  - ❖ Long acting effect due to slow release.
  - ❖ Prevent gluing of lashes.
  - ❖ Allows free exit of discharge.
- Systemic Ab.** (In severe cases).
- Dark glasses** for cases with photophobia.

**N.B.** NO bandage for cases having corneal ulcer secondary to MPC.

## 2-Purulent conjunctivitis

### Etiology:

- 60-80% of cases caused by: (Gonococci)**
  - Epidemics
  - Genital secretions → Contaminated hands →  
Infected discharge during delivery
- 20 % other organisms.**

### Clinical forms: Adult type:

- Incubation period:** Few hours to 3 days.
- Stage of infiltration:**
  - Eye lids:** (Tenderness- lid edema).
  - Conj.:** (Chemosis – hyperemia – No discharge but if presents it will be watery)
  - General signs:** (Fever, lymphadenopathy).
- Stage of discharge:** All of the previous decrease and large amount of purulent discharge appears.

### Fate and complications: Resolution within 2 weeks.

- Corneal ulcer (?? Why).
- Chronicity.
- I.C.



## Managment:

1- **Prevention:** (as MPC).

2- **Treatment:**

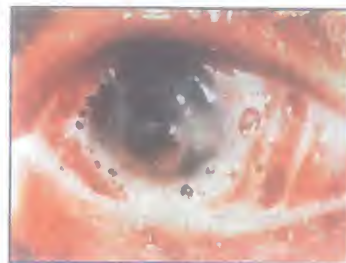
a) **Boric acid 4% lotion.**

b) **Topical antibiotic E.D.:** (broad spectrum Ab).

Dose: Every 5 minutes for an hour Then Every hour for 48 hours Then 4 times daily for 10 days

c) **E.oint by night**

d) **Systemic Ab:** single dose of Ceftriaxone.



## c) Ophthalmia neonatorum

**Definition:** Any conjunctivitis before or in the first month of life.

Any discharge from the eye of a new born infant is suspicious since tears are not secreted at this early date

### **Etiology:**

- 📌 Gonococcal (70% of cases).
- 📌 Other bacteria.
- 📌 Chlamydia.
- 📌 Viral infection.
- 📌 Chemical.



### **Clinical picture:**

**Symptoms:** given by the mother (Discharge – Red eye – Lid swelling)

**Signs:**

	Gonococcal	Other bacteria & Chlamydia	Viral	chemical
<b>Onset</b>	3-5 days	5 - 10 days	5-30 days	1 <sup>st</sup> day
<b>Discharge</b>	Purulent	Mucopurulent	Watery or mucoid	Serous
<b>Association</b>	Corneal	PAL	Dendritic ulcer Punctate Keratitis	lid edema is marked
<b>Investigation</b>	Culture & Sensitivity	❖ Culture & Sensitivity ❖ Immuno-fluorescent	Virology studies	
<b>Treatment</b>	Penicillin	Broad spectrum antibiotics	Antiviral	Stop E.D.



## d) Membranous conjunctivitis

**Causative organism:** Diphtheria

"Rare condition since children are now effectively immunized against diphtheria, Membranous conjunctivitis affects non-immunized children"

**Clinical picture:** Incubation Period: Few Hours to few days.

**Symptoms:** (Irritation – Red eye – Discharge → bloody purulent).

**Signs:**

1) **Systemic manifestations:**

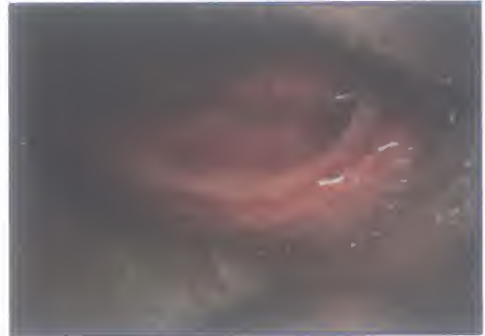
- a) Infection of the throat or the nasopharynx
- b) Constitutional symptoms (fever and malaise)

2) **Infiltration:**

- a) Eyelids → (edema & indurations) (Woody like).
- b) Conj. → (Chemosis & covered by true membrane).
- c) Systemic → P.A.L & fever manifestation.

3) **Discharge:** The membrane slough giving rise to bloody purulent discharge

**N.B.** True membrane is formed of (Discharge – Bacilli – Necrotic epith.)



**Complications:**

1) **Local complications** → due to healing by Fibrosis.

a) **Eyelid:**

- ❖ Entropion "Fibrosis of palpebral conjunctiva".
- ❖ Trichiasis or rubbing lashes.
- ❖ Symblepharon.

b) **Conjunctiva:**

- ❖ Xerosis: (Goblet cells atrophy - Obstruction of LG ducts – Accessory Lacrimal glands fibrosis).
- ❖ Conjunctival ulcers.

c) **Cornea:** (Marginal corneal ulcer – Xerosis – Vascularization).

d) **IC:** (Secondary to corneal ulcer).

2) **Systemic complications:**

- ❖ Toxic myocarditis → heart failure.
- ❖ Toxic nephritis.
- ❖ Respiratory failure.
- ❖ Toxic neuropathy.
- ❖ Optic neuropathy.
- ❖ Motor cranial nerves (3,4,5) → (paralytic squint).

### **Treatment:**

#### **1- Prophylaxis:**

- a) Immunization.
- b) Notification to healthy authorities and Isolation of sporadic cases.

#### **2- Actual treatment:**

##### **a) Systemic:**

- ❖ Anti-diphtheritic Serum (antitoxin)  
(40,000 - 60,000 U.) every 12 hours,
- ❖ Penicillin IM.
- ❖ Complete bed rest

##### **b) Local:**

- ❖ Anti-diphtheritic (antitoxin) Serum.
- ❖ Penicillin ED. Or broad spectrum E.D.
- ❖ Local Ab E.oint. (Placed using glass rod to avoid symblepharon).
- ❖ Atropine E.oint. (For corneal involvement).



## **e) Viral conjunctivitis**

### **Causative organisms:**

- 1) Adenovirus: type 8 or 19.
- 2) Herpes simplex & Enterovirus
- 3) Viral Fevers.

### **Clinical picture:**

#### **Symptoms:**

- 1) Irritation → discomfort.
- 2) Discharge → watery.
- 3) Red eye.

#### **Signs:**

- 1) Eyelid: Edema (vesicles).
- 2) Conj.: Edema (Chemosis) – Hyperemia – Sub conjunctival He – Conjunctival follicles.
- 3) Systemic manifestation: PLA
- 4) Corneal affection: Punctate Keratitis - Dendritic corneal ulcer.

**Fate:** Self limiting disease (resolve within one or two weeks).

**Treatment:** Antiviral drugs, e.g.: Acyclovir E.oint.



# Chronic infective conjunctivitis

## 1 - Chronic catarrhal inflammation

### **Etiology:**

- 1) **Sequelae of acute conjunctivitis.**
- 2) **General irritation:** dust, smoke, wind, heat.
- 3) **Local irritation:** rubbing lashes, PTDs.
- 4) **Errors of refraction.**

### **Clinical picture:**

- 1) **Irritation:** Discomfort, itching, burning and foreign body sensation.
- 2) **Little discharge.**
- 3) **Hyperemia of fornices.**

### **Treatment:**

- 1) Remove the cause.
- 2) Astringent and vasoconstrictor eye drops.

## 2 - Angular conjunctivitis

(See eyelid)

## 3 - Follicular conjunctivitis

**Definition:** Conjunctival inflammation characterized by follicle formation.

### **Causes:**

#### 1) **Viral infections:**

Adenovirus Herpes, Molluscum Contagiosum

- ❖ Follicles are more in the inferior fornix
- ❖ Cornea is free
- ❖ No scarring

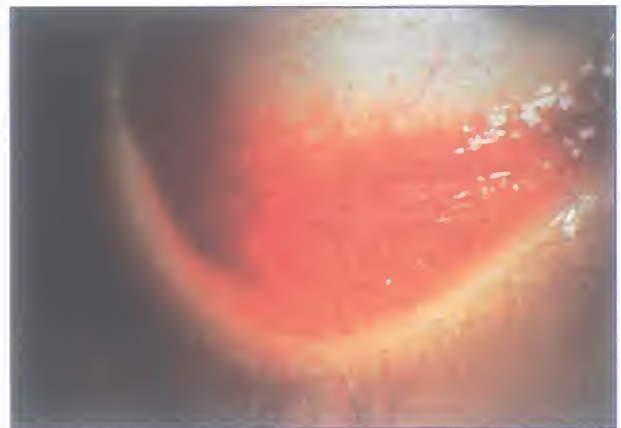
#### 2) **Chlamydia:**

Trachoma and adult inclusion conjunctivitis

#### 3) **Drugs (conjunctivitis medicamentosa):**

Atropine, Epinephrine, Glaucoma medications and Topical antibiotics

- ❖ History of drug use
- ❖ Itching
- ❖ Follicles are in the inferior fornix
- ❖ Rapid cure once the drug is stopped





#### 4) Folliculosis:

- ❖ Affects children
- ❖ Associated with enlarged tonsils or adenoids
- ❖ Follicles are arranged in parallel rows in the inferior palpebral conjunctiva
- ❖ Chronic course
- ❖ No scarring
- ❖ Cornea is free
- ❖ The condition improves by treatment of the cause

## 4- Trachoma

**Definition:** Chronic contagious inflammation of conj. due to infection by Chlamydia

trachomatis characterized by:

- 1) Formation of **follicles & papillae**.
- 2) **Pannus** formation.
- 3) Subepithelial **cellular infiltration**.
- 4) Healing by **fibrosis**.

1/3rd Endemic in Egypt (90% population) & 400,000,000 world wide

#### **Mode of infection:**

- ❖ Through conjunctival discharge carried by fingers, towels and flies
- ❖ Common in low socioeconomic areas and occurs at childhood in endemic areas

#### **Etiology:** (Chlamydia trachomatis):

#### Not virus:

- Size (↑ 200 µm) .
- Has cell wall (viruses DNA surrounded by protein cover).
- DNA & RNA.
- Sensitive to antibiotics.
- Do not give long lasting immunity.

#### Not bacteria:

- Obligatory intracellular.
- Produce intracytoplasmic inclusion bodies.

**Pathology:** Chlamydia → Epitheliotropic (conjunctival epith).

The granuloma enlarges and becomes raised above surface and undergoes centered necrosis  
And epithelium enlarges forming papillae at the same time

#### **Clinical picture:**

#### **Symptoms:**

- ❖ Irritation → Discomfort - F.B. Sensation.
- ❖ Red eye
- ❖ Mucoïd discharge - or mucopurulent discharge.

## Signs:

### 1) Conjunctival manifestations according to (MC Callens classification):

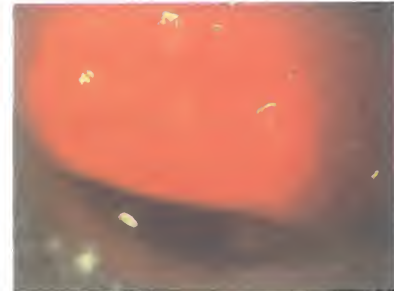
- a) **T1 → Minute follicles:** (Center of mononuclear cells surrounded by lymphocytes and giant multinucleated cells (Leber's cells).

- ❖ Small in size (11 mm) .
- ❖ Not raised above surface.
- ❖ Not expressible.



- b) **T2a → Large follicles:**

- ❖ Size 1-3 mm.
- ❖ Raised above surface,
- ❖ Expressible.



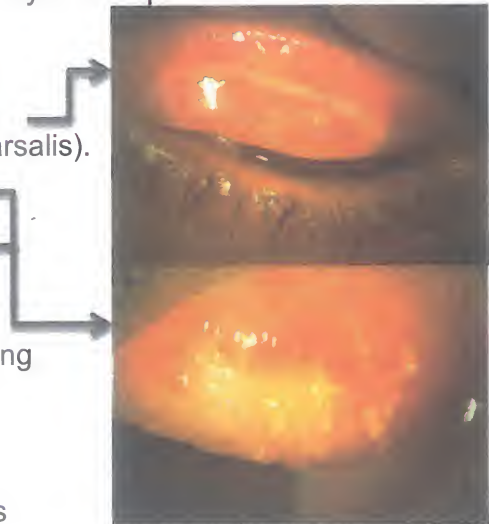
- c) **T2b → Papillae formation:**

- ❖ Finger like projection.
- ❖ Fine (small).
- ❖ Pink.
- ❖ Too many, (filling upper palpebral conj & fornix) may lead to ptosis.
- ❖ Giving a velvet appearance.

- d) **T3 → Fibrosis of palpebral conj (white patches):**

- ❖ Arlet's line (white line of Fibrosis in Sulcus sub-tarsalis).
- ❖ PTDS (Post trachomatous degenerations).
- ❖ PTCS (Post trachomatous calcifications).

- e) **T4:** As the previous stage but the patient is completely cured. (No I.B found on doing conj. scraping).

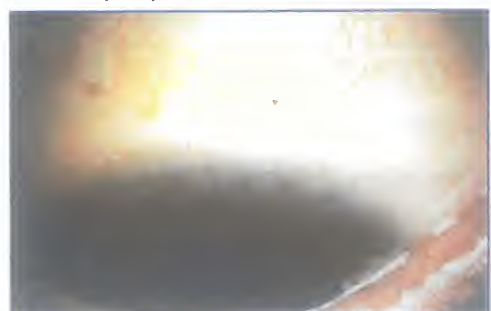
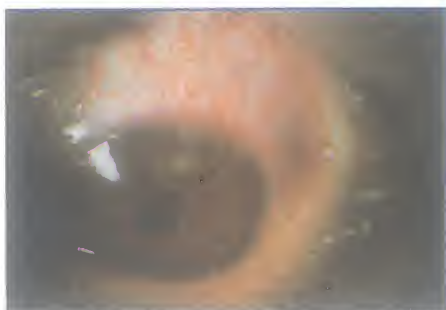


### 2) Corneal Signs:

- a) **Superficial Keratitis:** Numerous epithelial erosions involving the upper part of the cornea which shows positive staining with fluorescein

- b) **Corneal follicles: (called: Herbert's Rosette's):**

- ❖ Small greyish follicles at the upper cornea only at the end of limbal capillaries.
- ❖ Healing of these follicles leaves depressed pits (Herbert's pits).



### c) Pannus formation:

❖ **Definition:** Infiltration of the periphery of the cornea by chronic inflammatory cells & vascularization.

❖ **Clinical picture:**

- Progressive pannus: Infiltration precedes vascularization
- Regressive pannus: vascularization precedes infiltration which regresses.
- Healed - dry - pannus siccus: (Blood vessels changes to ghost vessels)  
Superficial and scar if B.M. is destroyed

❖ **Types of pannus:**

- Thick pannus → Pannus carnosus.
- Thin pannus → Pannus tennis.
- Vascular pannus → Pannus vasculosus
- Annular pannus → Pannus annulosus.
- Dry pannus → Pannus Siccus.



❖ **Fate of Pannus:** a Permanent opacity occur if Bowman's membrane is destroyed.

❖ **D.D.:** (see later).

### d) Trachomatous ulcer:

➤ **Typical:**

- ❖ At the lower end pannus.
- ❖ Horizontal - oval - superficial.

➤ **Atypical:** (any shape - any size - any where).

### e) Xerosis due to:

- ❖ Atrophy of goblet cells.
- ❖ Obstruction of L.G. ducts.
- ❖ Destruction of accessory L.G.

f) **Kerato ectasia:** (Bulging forward of weak cornea).

### **Complications:**

Eyelids	Conjunctiva	Lacrimal	Cornea
Cicatricial entropion.	Xerosis.	Dacryo-adenitis.	Pannus formation.
Trichiasis.	Symblepharon (eyelids).	Dacryo cystitis.	Corneal ulcer.
Chronic meibomianitis (chalazia).	Pigmentation.	Canaliculus.	Keratectasia.
<u>Ptosis:</u> (Mechanical) Fibrosis of Muller's Ms.	Hyaline and amyloid degeneration of conjunctiva & upper tarsus.	Punctual occlusion	Xerosis.



**D.D.:** Trachomatous pannus from other causes of pannus.

- ❖ Trachomatous papillae: from other causes of papillae mainly spring catarrh papillae.
- ❖ Follicles of trachoma: from other causes of follicular conjunctivitis.

### Diagnosis of Trachoma

#### 1) Clinical signs:

- ❖ Arlet's line.
- ❖ PTDs and PTCs.
- ❖ Pannus formation.
- ❖ Herbert's pits.

#### 2) Investigation:

Conj. scraping showing intra cytoplasmic inclusion bodies when stained with Giemsa stain.

### WHO classification of trachoma:

**TF: (trachomatous follicles):** detected in the lower fornix. F1 (insignificant), F2 (mild), F3 (moderate), F4 (severe).

**TI: (trachomatous inflammation):**

Severe diffuse inflammation obscuring 50% of large tarsal vessels.

**TS: (trachomatous scarring)** Grade 1, 2, 3.

**TT: (trachomatous trichiasis).**

**C.O.: (corneal opacification)** obscuring at least part of pupil with vision  $\frac{6}{18}$  or less

### Treatment:

#### Prophylaxis:

- ❖ Combat flies.
- ❖ Separate towels,
- ❖ Medications in both eyes.

#### Actual treatment:

- ❖ Systemic → Sulphadiazine tab. (1 gm/ t.d.s./ 10 days),
- ❖ Local → Sulphacetamide E.D & Terramycin E.oint.
- ❖ Atropine E.D. 1%.

#### Surgical treatment:

- ❖ Follicles → expression.
- ❖ Papillae → Scraping.
- ❖ PTDS, PTCS → Picking.

# Inclusion conjunctivitis

**Etiology:** Causative agent is chlamydia trachomatis type D transmitted from the genitals by fingers or through water of swimming pools (swimming pool conjunctivitis)

**Clinical picture:**

- 1) Lower lid follicles
- 2) Superficial punctate keratitis
- 3) Urethral or vaginal discharge
- 4) Spontaneous healing within 3 – 12 months

**Treatment:**

- 1) Sulfonamide or broad spectrum antibiotic eye drops and ointment
- 2) General sulfonamide, tetracycline or broad spectrum antibiotics



# Acute non-infective conjunctivitis

a) Mechanical injury: Dust, smoke, wind, physical or chemical agents.

b) Photophthalmia: (see cornea).

c) Acute allergic conjunctivitis:

**Etiology:** Type 1 hypersensitivity (to airborne plant or animal allergen)

**Clinical picture:**

- 1) Itching
- 2) Watery & Mucoid discharge.
- 3) Hyperemia.
- 4) Edema of lids.

**Treatment:**

- 1) Remove cause.
- 2) Astringent and VC drops.
- 3) Cold compresses
- 4) Artificial lubricants.
- 5) Non-steroidal anti-inflammatory drugs.
- 6) Anti-allergic
- 7) Topical steroids.



# Chronic non-infective conjunctivitis

## 1 - Phlyctenular conjunctivitis

**Definition:** Allergic conj. due to endogenous toxin as:

- ❖ Tonsillitis (exotoxin of streptococci).
- ❖ Chest T.B. (tubercle proteins).
- ❖ Intestinal infestation.
- ❖ Staphylococcal blepharoconjunctivitis

**Pathology:** Greyish white nodule formed of lymphocytic aggregation covered by intact epithelium which may ulcerate over the bulbar conjunctiva near the limbus or on the periphery of the cornea.

**Clinical picture:**

**Symptoms:**

**1) Phlyctenular conjunctivitis only:**

- a) Irritation → Discomfort & F.B. sensation.
- b) Watery discharge.

**2) Corneal affection:** Photophobia and lacrimation

**Signs:**

**1) Conj. Signs: (Nodule):**

- a) **Color:** Greyish white
- b) **Size:** 1 - 3 mm.
- c) **Site:** Bulbar conjunctiva near limbus.
- d) **No.:** May be multiple.
- e) **Surrounded by:** Dilated vessel.
- f) **Fate:** May ulcerate and 2<sup>ry</sup> Bacterial infection.

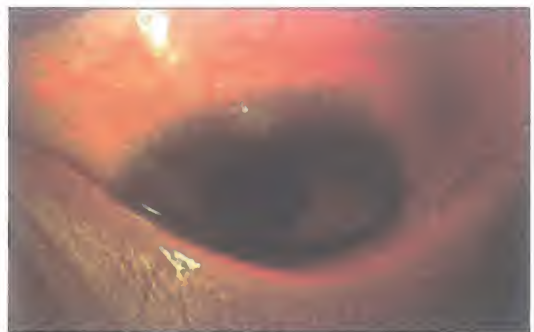
**2) Corneal manifestations:**

**a) Phlycten**

- ❖ Resolution.
- ❖ Vascularization giving rise to Phlyctenular pannus.
- ❖ Ulceration.

**b) Phlyctenular ulcer:**

- ❖ Marginal or peripheral ulcer.
- ❖ Annular ulcer (less common).
- ❖ Fascicular ulcer: Peripheral ulcer progress towards center in a serpiginous pattern and supplied by leash of blood vessels. (The ulcer leaves opacity Maximum when it stops).





N.B.: Serpiginous ulcer.

❖ Typical hypopyon ulcer.

❖ Mooren's ulcer

❖ Fascicular ulcer.

### Complications:

1) **Conj** → MPC (2<sup>ry</sup> bacterial infection).

2) **Corneal** → ulceration & opacification.

3) **Recurrence** → is common if cause is not treated.

**D.D.:**

1) **Phlycten must be differentiated from: (Pinguecula – epi-scleritis – Limbal spring catarrh)**

	Phlycten	Pinguecula
1- Age	Young	Old
2- Colour	Grey	Yellow
3- Shape	Rounded	Triangular
4- Site	Any where	Nasal side
5- Suppuration , ulceration & Vascularization	Present	Absent

	Phlycten	Episcleritis
1- Tenderness	Not tender	Tender
2- Age	Young	Old
3- Colour	Grey	Purple (deep vessels)
4- Level	Superficial	Deep
5- Movement	Moves with conjunctiva	Fixed to scleral (conj. moves Over it)
6- Adrenaline	Blanching (due to vasoconstriction of superficial bl. Vessels)	No blanching (deep blood. V.)
7- Ulc. & Supp.	Present	Absent

	Phlycten	Limbal spring catarrh
1- Itching	Absent	Present
2- Season	Any	Summer
3- Lesion	Nodular	Gelatinous masses
4- Ulc. & supp	Present	Absent
5- Oesinophils	Absent	Present

**2) Phlyctenular pannus must be differentiated from:**

	Trachomatous	Phlyctenular	Leprotic	Degenerative
1- Cause	Atypical virus	Allergy	Lepra bacilli	Absolute gl.
2- Site	Up	Anywhere		
3- Border	Serrated	irregular		
4- Vessels	Branching	Straight	Few Blood Vessels	

**TREATMENT:**

- 1) Cortisone E.D& E. Oint.
- 2) Dark glass.
- 3) Treatment of the cause.
- 4) Treatment of fascicular ulcer by Actual cautery using red hot platinum needle.  
For the leash of vessels, chemical cautery by carbolic acid for the ulcer.
- 5) Local atropine E Oint if cornea is involved.

## 2- Spring catarrh (Vernal Keratoconjunctivitis)

**Definition:** Bilateral, Recurrent, seasonal allergic conjunctivitis due to allergy to an exogenous allergen such as: pollen grains, dust, fumes and U.V. rays.

**Incidence:**

- ❖ More Common in boys.
- ❖ Associations (Keratoconus - Contact lens wearers).

**Clinical picture:**

Symptoms:

- ❖ Irritation: F.B - Discomfort - Itching,
- ❖ Red eye.
- ❖ Discharge: Thready discharge.

Signs:

1) Conjunctiva:

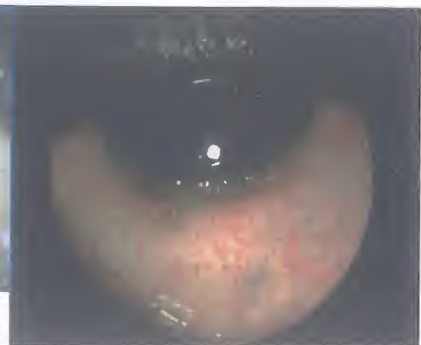
a) Palpebral type (Papillae):

- ❖ Large.
- ❖ Flat topped.
- ❖ Bluish-white or red
- ❖ Fornix free.
- ❖ **Cobble stone arrangement.**
  - ❖ On eversion of eyelid for one minute it will be covered by **milky sticky fluid rich in eosinophils.**
  - ❖ Formed of a **central core of fibrous tissue** rich in **eosinophils** and covered by **thick epithelium**

b) Bulbar type:

- ❖ **Gelatinous masses** starts around the limbus then later all around.
- ❖ **Tranta's Spots:** White spots within gelatinous masses. (Represents necrotic epith.).
- ❖ Formed of **hypertrophied epithelium** with **connective tissue core** and **Hyaline degeneration**

c) Mixed type.





## 2) Cornea:

- a) S.P. Epitheliopathy.
- b) Superficial C.U.
- c) Annular (360) pannus.

Stained with fluorescein



### Treatment:

- 1) **Dark glasses.**
- 2) **Cold compresses** to induce V.C. → ↓ blood flow → ↓ release of inflammatory mediators.
- 3) **Antihistaminic E.D.** e.g.: Livostin & Nostamine.
- 4) **Vasoconstrictive E.D.** (decongestant E.D.) e.g.: Visine & Prisoline.
- 5) **Mast Cell stabilizers:**
  - a) Disodium chromoglycate (Optichrome E.D.)
  - b) Lodoxamamid (Epichrome E.D.).
- 6) **Change Surrounding atmosphere.**
- 7) **NO improvement:**
  - a) Corticosteroid E.D. and E.Oint.
  - b) Intra-papillary injection cortisone.
  - c) B. irradiation (endarteritis-obliterans) applications,
  - d) Immuno-suppressive therapy (cyclosporin).

## 3- Giant papillary conjunctivitis

### Etiology:

- 1) Contact lenses wear especially extended-wear contact lenses.
- 2) Irritation with ocular prosthesis or by exposed corneal sutures.

### Clinical picture:

#### Symptoms:

- 1) Contact lens intolerance.
- 2) 2<sup>ry</sup> Irritation: discomfort, burning and foreign body sensation.
- 3) Watery or Mucoïd discharge.
- 4) Hyperemia.



**Signs:** Affects the superior tarsal conjunctiva. They are flat-topped covered with mucous

### Treatment:

- 1) Discontinuation of use of offending lens (Symptoms resolve but papillae will persist for several months)
- 2) Topical treatment as in spring catarrh.

# Degeneration of conjunctiva

## 1 - Pinguecula

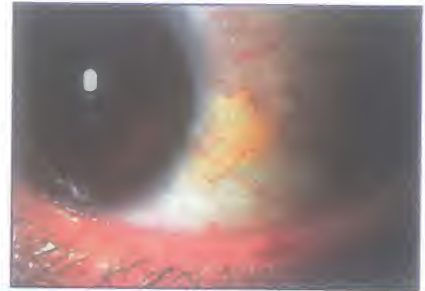
**Definition:** Degenerative condition of conj. in old age with yellow, raised, triangular nodule, nasal to limbus.

**Pathology:** Area of hyaline degeneration with deposition of elastic tissue due to U.V. rays exposure.

**D.D.:** From Phlycten.

**Treatment:**

- + No tt
- + Excision: if large & disfiguring.



## 2 - Pterygium

**Definition:** It is triangular encroachment of conjunctiva over cornea.

AE: Unknown Ppfs:

- ❖ **Chronic irritation** as: Sun (Ultraviolet rays), heat, dust.
- ❖ **Pinguecula** (Not certain).

**Pathology:**

- ❖ Thin layer of conj. Epith.
- ❖ Fibro-vascular membrane (Fibrous tissue & Bl. vessels).
- ❖ Bowman's membrane & superficial layers of stroma are destroyed.

**Clinical picture:**

**Symptoms:** 3 D

- + Disfigurement.
- + Diminution of vision (if cross pupil) & irregular astigmatism.
- + Diplopia (rare) d.t. limited movements.

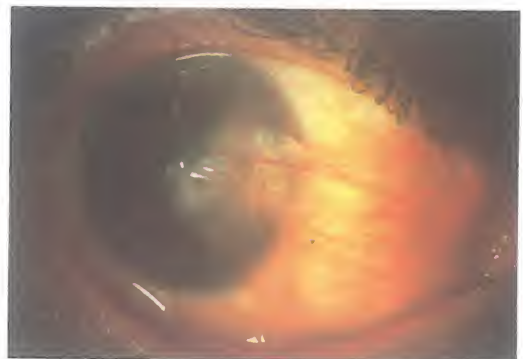
**Signs:** A triangular fold commonly nasal and bilateral.

It consists of:

- 1) **Apex:** over cornea.
- 2) **Neck:** at limbus.
- 3) **Body:** over sclera.

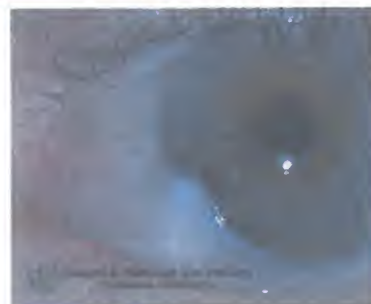
**Types:**

- 1) **Regressive:** Thin (Membranous) less vascular, it creeps towards the center of the cornea
- 2) **Progressive:** Thick (Fleshy) more vascular.



**D.D.:**

	<b>True Pterygium</b>	<b>Pseudo Pterygium</b>
<b>Nature</b>	Degenerative condition Unknown AE	A fold of conj. attached to base of healed ulcer
<b>Site</b>	Commonly nasal	Any where
<b>Side</b>	Commonly bilateral	Unilateral
<b>Hook</b>	Cannot pass under neck	Can pass
<b>Course</b>	Stationary or progressively	Stationary



**Treatment:**

1) Follow up: If Pterygium is small & stationary as recurrence is common after operation.

2) Operative treatment:

a) Indication:

- ❖ **Progressive Pterygium** with encroachment on pupil or disfigurement.
- ❖ **Recurrent Pterygium.**
- ❖ **Disfigurement** annoying patient.

✚ Excision with bare sclera (D'Ombrian) operation:

Pterygium is dissected & excised leaving small bare area of sclera near limbus (To allow the cornea to be covered by corneal epith. not conj. Epith.).

✚ Excision with grafting: (especially in recurrent cases):

Excision of Pterygium + covering the raw area by:

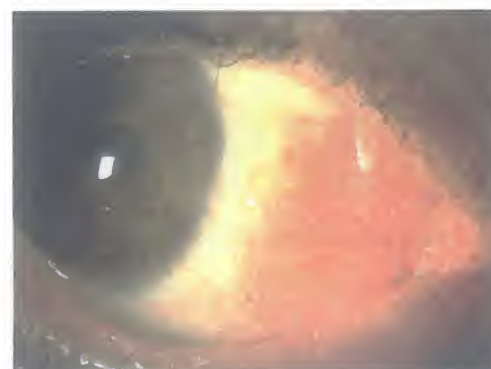
- Conjunctiva.
- Amniotic membrane graft.
- Corneal graft (keratoplasty).

3) Postoperative treatment: (to prevent recurrence):

a) **Beta irradiation:** inhibits Blood vessels  
(produce endarteritis-obliterans).

✚ Dose: 2500 Rads by strontium 90.

b) **Mitomycin C.**



**Pterygium after excission**



# Dry eye

## Etiology:

- 1) Idiopathic atrophy of the main and accessory lacrimal glands
- 2) Congenital absence of the lacrimal gland.
- 3) Inflammation of lacrimal gland e.g. sarcoidosis.
- 4) Tumors of lacrimal gland e.g. mixed lacrimal gland tumor.
- 5) **Kerato-conjunctivitis sicca**: autoimmune disease leading to atrophy and fibrosis of the lacrimal gland, it occurs usually in females and may be associated with arthritis and dry mouth (**Sjogren's syndrome**).



- 6) **Conjunctival scarring**: due to trachoma, chemical burns, Stevens Johnson syndrome.
- 7) **Medications that suppress tear secretions such as**: Anticholinergics and Antihistaminics
- 8) **Xerosis**: this is a condition where the conjunctiva becomes thickened, and loses its luster and transparency.
  - ✚ The epithelium may become keratinized, causing white foamy patches known as bitot spot.
  - ✚ In severe cases, scarring produces symblepharon.
  - ✚ Tear deficiency occurs due to loss of goblet cells and accessory lacrimal glands and obstruction of the ducts of the main lacrimal gland
  - ✚ **Causes of xerosis include** Vitamin A deficiency , post-inflammatory scarring as a sequel of trachoma, chemical and thermal burns, Stevens-Johnson syndrome, Ocular pemphigoid, irradiation and lagophthalmous

## Clinical picture:

**Symptoms:** Irritation and foreign body sensation.

**Signs:**

- 1) Deficient pre-corneal tear film.
- 2) Punctate epithelial erosion of the cornea.



### Investigations:

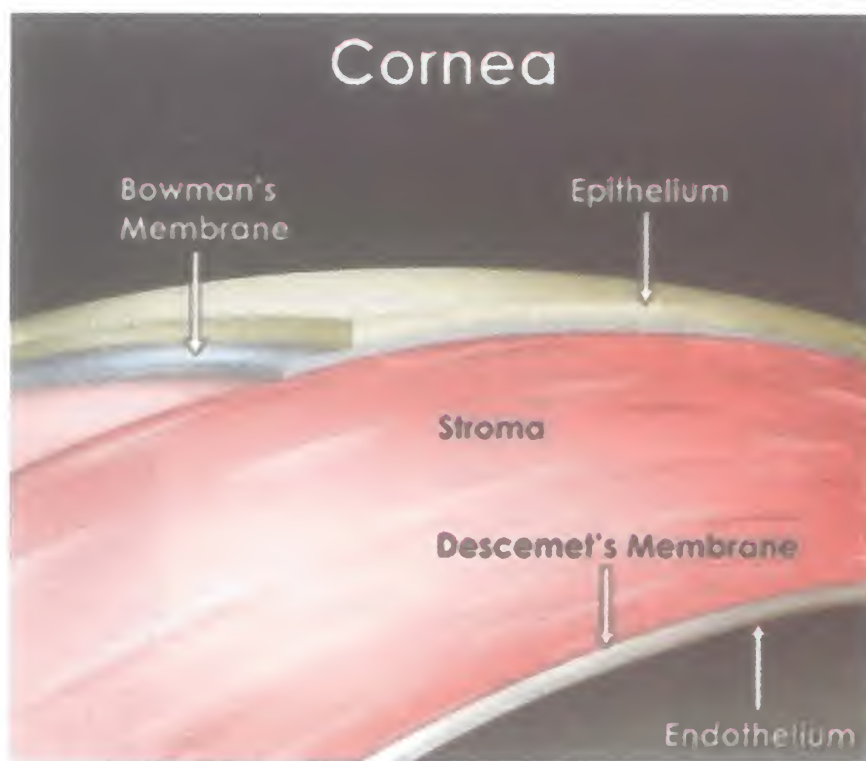
- 1) Tear film break - up time (BUT): is diminished (normally it is 15 seconds).
- 2) Schirmer's test: a normal person wets 10-30 mm of a Whatman number 41 filter paper strip (5 mm. wide X 30 mm. long) in 5 minutes. Values less than 5 mm indicate hypo-secretion. →
- 3) Rose Bengal staining: of devitalized epithelial cells.

### Treatment:

- 1) Protective glasses and contact lenses.
- 2) Artificial tears eye drops.
- 3) Occlusion of the puncti to reduce tear drainage.
- 4) Systemic steroids.



# Cornea





# Cornea

- ❖ It is the anterior  $\frac{1}{6}$  of the outer coat.
- ❖ It is transparent and avascular except 1 mm at the limbus.
- ❖ It fits into the anterior scleral foramen as a watch glass.

## Anatomy

### Gross Anatomy:

#### Corneal diameter:

- ❖ Vertical diameter **11 mm** (10 mm in infants).
- ❖ Horizontal diameter **12 mm** (11 mm in infants)

**Note:** Vertical diameter is less by one centimeter

due to conjunctival overlap.

#### Corneal thickness:

- ❖ At the center → **0.55 - 0.6 mm**.
- ❖ At the limbus → **from 0.67 up to one mm**.

**Corneal curvature:** Radius of curvature is about 7.8 mm

### Microscopic Anatomy:

#### 1) Epithelium: 5-6 layers of stratified Squamous epithelium (Non Keratinized):

- ❖ One layer of deep basal columnar cells lying on a basement membrane.
- ❖ Their nuclei are oval with their long axis perpendicular to the basement membrane
- ❖ Two or three rows of wing shaped cells with their nuclei parallel to the surface,
- ❖ Two rows of flattened squamous non-Keratinized cells on the surface.

#### 2) Bowman's membrane:

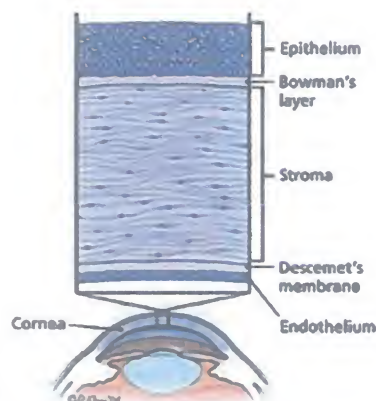
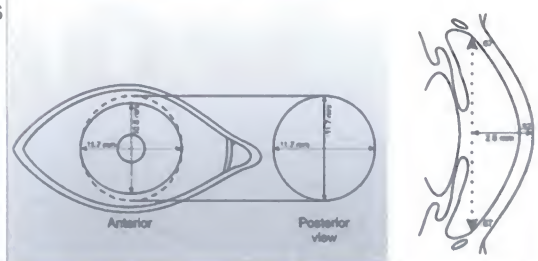
- ❖ Superficial condensed layer of the stroma.
- ❖ Elastic membrane. ❖ Acellular.
- ❖ Incapable of regeneration.

#### 3) Stroma:

- ❖ Represents **90% of corneal thickness**.
- ❖ Formed of **about 100 regularly arranged layers of collagen fibers** arranged in bundles which are:

- ✓ Closely packed together.
- ✓ Runs parallel to the corneal surface.
- ✓ Cross each other perpendicularly.
- ✓ Bathed in mucopolysaccharide and lymph.
- ✓ Peripherally there are spaces which contain fixed cells (Keratocytes) and wandering cells (macrophages).

### Corneal Dimensions



#### 4) Descemet's membrane:

- ❖ Elastic membrane.
- ❖ The basement membrane of the endothelium (cellular).
- ❖ Capable of regeneration.

#### 5) Endothelium:

- ❖ Single layer of flat hexagonal endothelium cells.
- ❖ **Function:** pumping out of aqueous (corneal dehydration).
- ❖ **Incapable for regeneration**, when damaged neighboring cells enlarge to fill in the spaces.
- ❖ **Count of Endothelium is: (4 – 4000 cell/mm<sup>2</sup>)** least count to keep its function is 400 cell/mm<sup>2</sup>.

**The limbus:** It is an important landmark in eye surgery. Automatically, this is a zone between the cornea from one side and conjunctiva and sclera from the other side. At the limbus the following changes occurs.

- 1) **Corneal epithelium** becomes continuous with conjunctival epithelium.
- 2) **Bowman's membrane** ends in a round border, short distance from the limbus.
- 3) **Stroma** becomes continuous with the sclera.
- 4) **Descemet's membrane** ends in a rounded border known as Schwalb's line.
- 5) **Corneal endothelium** becomes continuous e T.M and iris endothelium.

Refractive power is 42 D - 45 D  
Refractive index is 1.37

#### **Nutrition of the cornea:**

By diffusion from: (Tears – Aqueous humor – Limbal capillaries).

#### **Nerve Supply:**

- ❖ **Trigeminal nerve → Ophthalmic division → Naso ciliary nerve → long ciliary nerves.**
- ❖ These long ciliary nerves from a rich.
  - Stromal Plexus.
  - Sub-epithelial Plexus.
  - Intraepithelial Plexus.

N.B 1: The nerve endings of the cornea have the least threshold of pain

N.B 2: Nerves are non-myelinated

### Causes of corneal transparency:

- 1) **Epithelium:** (non-keratinized) & covered by tear film.
- 2) **Stroma:**
  - a) Regular arrangement of collagen Bundles.
  - b) Nerves: Non-myelinated.
  - c) Vessels: Avascular.
- 3) **Endothelium:** Endothelial pump to keep cornea dehydrated.

**Corneal opacification:** Disturbance of any of the above mentioned factors could result in corneal opacification

- ❖ **Surface irregularity, dryness and keratinization of the epithelium** can disturb the corneal clarity
- ❖ **Irregularity of the stromal arrangement** usually follows corneal scarring
- ❖ **Abnormal stromal contents** such as blood vessels, inflammatory cells, edema and mucopolysaccharides all lead to loss of clarity
- ❖ **Endothelial damage (failure of endothelial pump)** will lead to severe corneal edema and marked loss of clarity.

### Function of the cornea:

- 1) Main refractive surface of the eye.
- 2) Protective function through corneal sensation.

## Corneal diseases

### Definition of corneal lesions:

- 1) **Corneal ulcer:** loss of corneal epithelium and superficial layers of the stroma.  
May be infective or non infective.
- 2) **Corneal abrasion:** loss of corneal epithelium due to mechanical trauma.
- 3) **Keratitis:** inflammation of the cornea. May be ulcerative or non-ulcerative, infective or non-infective.
  - a) **Superficial keratitis:** inflammation of epithelium and superficial stroma.
  - b) **Interstitial keratitis:** inflammation of stroma.
  - c) **Superficial punctate keratitis:** Dot shaped inflammation & infiltration.
    - ❖ **Etiology:** viral, drug induced, dryness and bacterial toxins.
- 4) **Superficial punctate erosions:** Dot shaped superficial epithelial defects.
- 5) **Corneal haze:** loss of corneal clarity due to edema or infiltration.
- 6) **Cornel scar:** loss of corneal clarity due to replacement of regular stroma by irregular fibrous tissue after healing of a corneal pathology. Classified as:
  - a) **Nebula** (see later).
  - b) **Leucoma adherent** (see later).
  - c) **Leucoma** (see later),
  - d) **Descematocele** (see later).



# Keratitis

**Definition:** Inflammation of the cornea, which may be infective or non infective.

**Classified into:**

- 1) **Superficial Keratitis:** Inflammation involves epithelium and sup. stroma which may be:
  - a) Ulcerative (corneal ulcer).
  - b) Non-ulcerative.
- 2) **Interstitial Keratitis:** Inflammation involves stroma with intact epithelium

## Corneal protective mechanisms against C.U.:

1) **Eye lids:**

- ❖ Closure during sleep protects the cornea from dryness and exposure
- ❖ Tears distribution.

2) **Tear film:**

- ❖ Protect cornea against dryness and ulceration
- ❖ Contains lysozymes that have antimicrobial effect.

3) **Corneal sensation:** Irritation of the cornea causes Reflex blinking and lacrimation.

4) **Intact corneal epithelium protects cornea against all micro organisms except:**

- ❖ N.Gonorrhea.
- ❖ C.Diphtheria.
- ❖ H.Egypticus.
- ❖ Listeria.

# Corneal ulcer

**Types of corneal ulcer:**

1) **Non-infective corneal ulcer.**

2) **Infective C.U.:**

- |               |               |
|---------------|---------------|
| a) Bacterial. | c) Fungal.    |
| b) Viral.     | d) Protozoal. |

# Bacterial corneal ulcer

**Etiology:**

1) **predisposing Factors:**

a) **General predisposing Factors (↓ Immunity):**

- ❖ DM.
- ❖ Old age.
- ❖ Vitamin A & C deficiency.
- ❖ Malnutrition.

**b) Local predisposing Factors:**

- ❖ Lagophthalmos (exposure).
- ❖ Dry eye (Xerosis).
- ❖ Loss of corneal sensation.
- ❖ Loss of intact corneal epith. (Abrasion).

**2) Causative organism:**

- a) The 4 virulent organisms.
- b) **Pseudomonas** (Cause C.U. in contact Lens wearers).
- c) **Pneumococci** (cause hypopyon ulcer).

**3) Source of infection:**

- a) Blepharitis.
- b) Dacryocystitis.
- c) Conjunctivitis.
- d) Contact lens.

**Pathology of C.U.:**

**1) Stage of infiltration (progressive stage):** The organism invades the cornea causing:

- a) Localized area of Necrosis (appears as gray area).
- b) Acute inflammatory cells (PNL) from limbal capillaries migrate and surround the necrotic area.

**2) Stage of ulceration:**

- ❖ Part of the necrotic tissue slough causing an ulcer which has the following characters:  
(Shallow ulcer – Irregular – Greyish in color).
- ❖ The fate then depends upon the response to treatment or tissue resistance (immunity) against the virulence of the organism:

**If the immunity is stronger then we are in regressive (stage of clean ulcer)**

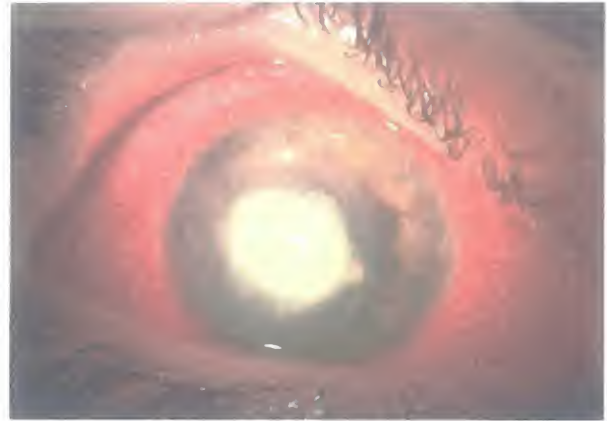
this is because macrophages engulf the necrotic tissue.

(Deep – Regular base – More transparent).

**If the organism is very virulent then we still stage of perforation.**

**3) Stage of healing:**

- a) Stroma & Bowman's heals by fibrosis or fibrosis & vascularization.
- b) Epithelium heals by epithelization (Mitosis) & sliding epith.  
(Cells at the edge of the ulcer).



### **Clinical picture:**

#### **Symptoms:**

##### **1) Pain:**

- ❖ Character: severe & stitching.
- ❖ Cause: Irritation of exposed nerves by toxins & eyelids blinking.

##### **2) Blepharo spasm.**

##### **3) Photophobia.**

##### **4) Lacrimation.**

##### **5) Visual acuity:** Area of the ulcer causes opaque area in the cornea → Associated uveitis.

##### **6) Red eye.**

#### **Signs:**

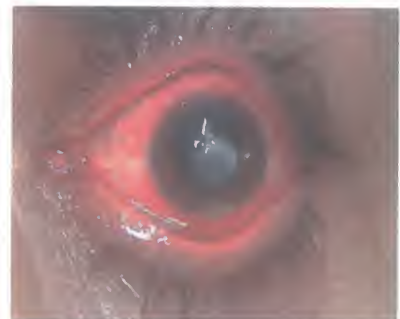
##### **1) Lid edema** (due to Blepharospasm).

##### **2) Conjunctiva shows ciliary flush.**

##### **3) Cornea:**

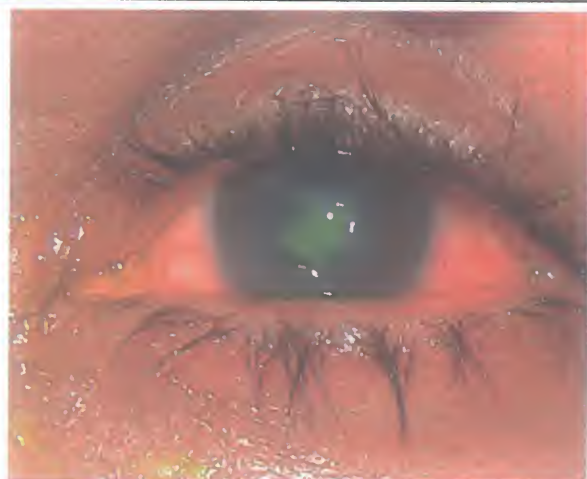
- Grey area.
- Loss of luster (due to loss of intact epith at site of ulcer).
- (+ve) fluorescein test (1%) (Diagnostic test).

##### **4) Iris:** Signs of iritis (Flare – Cells – Hypopyon)



### **Technique of fluorescein test is:**

- 1) Instill a drop of fluorescein eye drops (yellowish in color)
- 2) Wait for a minute and then wash the fluorescein eye drops with saline.
- 3) Examine the cornea using blue cobalt filter added to site of the ulcer will appear to have greenish coloration (stained with fluorescein)





## Complications (V. Imp.):

### 1) Non-perforating C.U.:

- a) **Secondary Iridocyclitis:** Due to diffusion of toxins from the site of ulcer (sterile I.C.).
- b) **2ry glaucoma:** Due to plasmoid aqueous.
- c) **Descematocele:** It is herniation of Descemet's membrane at the base of an ulcer in front of high I.O.P.
  - ❖ Clinically: appears as a clear vesicle at the base of the C.U.
  - ❖ Fate: either rupture (perforation occurs) or healing occurs.
  - ❖ Rare in: Children (weak descemet's) & Hypopyon ulcer (post. abscess formation)

### d) Complications as a result of healing:

#### ✚ Typical corneal opacities:

- ❖ Types → Faint superficial Opacity (Nebula).
  - Deep dense corneal opacity (leucoma).
  - Medium opacity (Macula).
- ❖ Causes: ↓ V/A either directly (if central) or indirectly through making corneal surface irregular causing astigmatism.



#### ✚ Atypical corneal opacities:

- ❖ Facet: Depressed corneal scar lined by epithelium (Not stained with fluorescein).
- ❖ Kerectasia: Ectasia of weak corneal scar.
- ❖ Pseudo-Pterygium: Fold of conjunctiva adherent to ulcer base.

### 2) Complications of perforating corneal ulcers:

#### a) Peripheral perforation:

- ❖ Leads to iris prolapse causing leucoma adherent (Dens opacity to which iris is adherent).
- ❖ Ectasia of the leucoma adherent may occur in front of high IOP and then it is known as: (Anterior Staphyloma).

**N.B. : Leucoma adherent may lead to 2ry angle closure glaucoma.**

#### b) Central perforation:

##### ✚ Small central perforation:

- ❖ Healing of central perforation is so difficult as the iris will not reach site of perforation and blood vessels will not reach the extreme center of the cornea, therefore healing will depend on fixed keratocytes.
- ❖ **Fixed keratocytes will form a fibrin plug which may:**
  - Remain and changes into fibrous tissue (leucoma non adherent).
  - Dislodge in front of ↑ IOP → Then the epithelium will descend to line track of perforation (Fistula).

❖ **Complications:**

- Infections.
- Anterior polar cataract.
- Hypotony (causes macular edema).

**Diagnosis of central perforation**

1) Flat or lost A.C.

2) Soft eye.

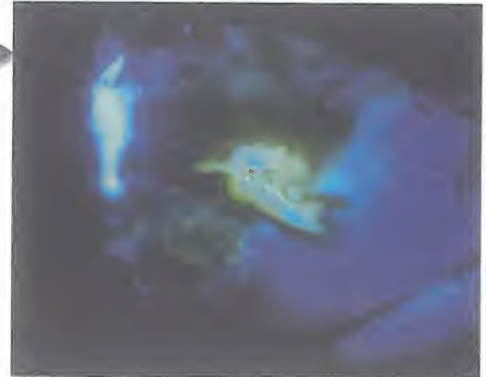
3) Seidle test will be positive.

**Seidle test**

- ❖ Instill a drop of fluorescein and examine the eye using blue light of the slit lamp.
- ❖ Press on the globe.
- ❖ The coming out aqueous through perforation site will dilute the fluorescein stain.

✚ **Large central perforation: Causes:**

- ❖ Infections.
- ❖ Expulsive Hemorrhage.
- ❖ Subluxation and dislocation of the lens  
(as a result of sudden negative pressure).



3) **Other complications:**

- a) **Complicated cataract**
- b) **Endophthalmitis as a complication of perforation**
- c) **Hypotony**

**Causes of rapid progression and early perforation of a corneal ulcer**

- 1) **Low immunity:** in diabetics, in association with local and systemic steroid use and in patients on immunosuppressive treatment
- 2) **Virulent organisms:** such as beta hemolytic streptococci and gram -ve bacilli (Pseudomonas)

**Signs of perforation of a corneal ulcer**

- 1) **Pain relief** (due to reduction of intraocular pressure)
- 2) **Soft tension** (digitally)
- 3) **Flat anterior chamber**
- 4) **Seidel's test (fluorescein test):** escape of aqueous through the perforation is seen as a green stream with gentle pressure on the globe after instillation of fluorescein eye drops

	Leucoma non-adherent	Leucoma adherent
<b>Cause</b>	❖ Healing of non perforating C.U. ❖ Healing of centrally perforating C.U.	Healing of peripheral perforated C.U.
<b>A.C.</b>	Regular depth	Irregular depth
<b>Pupil</b>	Rounded and regular	Peaked or pear shaped
<b>IOP</b>	Normal IOP	May be increased
<b>Color</b>	Whitish corneal opacity	Pigment corneal opacity

### Grading of corneal ulcer

	Size	Depth (%)	Sclera
<b>Mild</b>	↓ 2 mm	↓ 20 %	Not affected
<b>Moderate</b>	2.5 mm	20 – 50 %	Not affected
<b>Severe</b>	↑ 5 mm	↑ 50 %	Affected

**Investigations:** (Laboratory work up):

**1) Culture & smears:** Taken from the lids & conjunctiva of both eyes as well as corneal scrapings from the bed and edges of ulcer.

**2) Culture & sensitivity test:** Show antibiotics that were effective in killing the organism.

**N.B.1:** The test may take few days, so we don't wait for results but **we should start treatment immediately by broad spectrum antibiotics** (cover gram +ve & -ve organisms).

**N.B.2:** **Negative culture doesn't rule out an infective etiology of the ulcer** because topical antibiotics can clean the ocular surface from pathogenic organisms but still present in the corneal stroma.

**3) Corneal biopsy: (Indications):**

- Fungal or Acanthamoeba keratitis.
- Collagen or metabolic disease.
- Resistant ulcers (not improved with treatment).



## Treatment of C.U.:

### 1) Topical treatment:

#### a) Antibiotic Eye drops and Eye ointment:

- ✦ **Mono-therapy Broad spectrum antibiotics (Fluoro-quinolones) e.g.: ciprofloxacin E.D. (0.3%)** (covers almost all organisms except some species of staph aureus and pseudomonas).
  - ❖ The new fourth generations Fluoro-quinolones (as moxifloxacin 0.5% & gatifloxacin 0.3%) are more popular in use.

Or

#### ✦ Fortified or concentrated E.D:

- ❖ Fortified amino-glycosides: fortified Garamycin or Tobramycin 15 mg/ml (against gram -ve organisms) and amikacin 10mg/ml (against gram -ve bacilli & resistant staph).
- ❖ Fortified cephalosporins: (fortified vancomycin orcefobid 50 mg/ml) (against gram + ve organisms).
- ❖ Dose of Antibiotic therapy: Either →  
Every 5 minutes 5 times then every 30 minutes for 24 hours then 5 times daily.  
OR Every 5 minutes 5 times then 5 times daily.

**N.B. Sometimes Antibiotics may be given sub-conjunctival**

### Failure of response to antibiotics E.D may be due to:

- 1) Wrong diagnosis: (Non infective C.U. ---- Non-bacterial organism).
- 2) Wrong choice of antibiotic.
- 3) Drug toxicity (antibiotic is toxic on corneal epith).
- 4) Immuno-compromised patients due to systemic disease or drug-induced.
- 5) Persistent pre-disposing factors.

#### b) Cycloplegic E.D: (See uveitis)

### N.B.:

- ❖ **Atropine is better given as an eye ointment in children** to decrease systemic absorption.
- ❖ **In atropine sensitivity** we give cyclopentolate 1%
- ❖ **If pupil resist dilatation**, give sub-conjunctival injection of mydracaine:
  - Atropine 1%
  - Adrenaline  $\frac{1}{1000}$
  - Novocaine 2%

c) Eye patching:

- ❖ Promotes epithelial healing through avoiding blinking,
- ❖ Prevent photophobia.
- ❖ Contraindicated in: presence of conjunctivitis with discharge.

d) Bandage soft C.L.: Collagen shields may be impregnated with antibiotics.

**2) Systemic treatment:**

a) Systemic Antibiotics: Indicated if:

- ❖ Ulcer near limbus
- ❖ Sclera is involved.
- ❖ Ulcer is perforated. (Otherwise it will be of no benefit as the cornea is avascular).

b) NSAIDS.

**3) Treatment of the causes:**

- ❖ Remove: PTDS & Rubbing lashes.
- ❖ Stop wearing contact lenses.
- ❖ Control DM.

**4) Surgical Treatment:**

a) Cauterization (to kill organism): Better by using carbolic acid as it penetrates the ulcer deeply and harmless to the rest of the cornea.

b) Paracentesis: (Aspiration of aqueous using insulin syringe).

- ❖ ↓ IOP.
- ❖ Wash out toxins.
- ❖ New aqueous is formed rich in Antibodies & inflammatory cells.
- ❖ Indications: (Impending perforation --- Hypopyon and secondary glaucoma).

c) Conjunctival flap: (Flap of conj is dissed and used to cover the ulcer and fixed with suture to the limbus).

- ❖ Value:
  - Prevent perforation & promote healing of the ulcer being vascular.
  - ↓ Complications of perforation and help healing of the perforation track.

d) Contact lens: (therapeutic C.L or T. lens)

- ❖ Prevent perforation.
- ❖ ↓ Risk of small perforation.

e) Tissue adhesive glue: (cyanoacrylate)

f) Tarsorrhaphy: (suturing lid margins together)

- ❖ Lateral tarsorrhaphy.
- ❖ Median tarsorrhaphy.

g) Therapeutic Keratoplasty:

### 5) Signs of improvement:

- a) ↓ Area of ulcer.
- b) ↓ Depth of ulcer.
- c) Improvement of symptoms.

### 6) Treatment of complications:

- a) Iritis: Never ttt by steroids as it retards healing & leads to flare up of infection. Instead give NSAIDS.
- b) 2<sup>ry</sup> glaucoma: medical treatment & surgical (Paracentesis).
- c) Descematocele:
  - ❖ Hospitalization.
  - ❖ Medical ttt to lower IOP.
  - ❖ Surgical ttt. (Paracentesis - conj flap - C.L. - Keratoplasty).
- d) Perforation:
  - ❖ Hospitalization.
  - ❖ Small perforation (Tissue glue – C.L.).
  - ❖ Large perforation (conj flap – Keratoplasty).
- e) Fistula:
  - ❖ Destruction of epith. tract by red hot platinum needle then treat as perforated ulcer.

## Acute serpiginous ulcer (Hypopyon ulcer)

**Definition:** Disc shaped ulcer associated with severe I.C. & hypopyon.

**Etiology:** As bacterial C.U.

- ❖ Organism → Typical hypopyon ulcer Caused by pneumococci (80%).



- ❖ Atypical hypopyon ulcer Caused by other organisms as (Staph – Strept).

### **Pathology:**

- ❖ Stage of infiltration.
- ❖ Stage of ulceration.
- ❖ Stage of healing.

### The typical hypopyon ulcer has the following features:

- 1) Ulcer shows a serpiginous pattern as it spread towards the centre.
- 2) It has:
  - a) Healing edge: (Sloping – Near limbus – Little infiltration around it – Epithelialized).
  - b) Advancing edge: (Towards the centre – Undermined – Surrounded by dense infiltration).



### **Clinical picture:**

**Symptoms:** as bact. C.U. (Pain – Blepharospasm – Photophobia – Lacrimation – V.A.)

**Signs:** as before.

**Complications:** As before but:

- 1) I.C is severe and shows hypopyon & glaucoma.
- 2) Descematocele is rare due to posterior abscess formation.
- 3) Perforation is common → Posterior abscess formation → spread towards the center which is thinner than the periphery of the cornea.

**Treatment:** as before

## FUNGAL C.U.

### **Etiology:**

- 1) **General predisposing factors:** (as before).
- 2) **Local predisposing factors:**
  - a) Trauma with objects of plant origin.
  - b) Improper use of steroids.
- 3) **Organism:** Fungus e.g.: (Asperigellus).

**Clinical picture:** Characteristic features:

- ❖ Dry grey ulcer.
- ❖ Stromal infiltration with feathery edges and satellite lesions.
- ❖ Endothelial plaque.
- ❖ Thick cheesy hypopyon.

### **Investigations:**

- ❖ Cultures on specific media.
- ❖ Corneal biopsy.
- ❖ Confocal scanning microscope.

### **Treatment:**

- 1) **Topical Antifungal (have poor corneal penetration): (Amphotercin B):**

(Nystatin – Fluconazole – Natamycin – Miconazole – Ketoconazole – Itraconazole)

- 2) **Systemic Antifungal:** as: (Itraconazole – Ketoconazole).

- 3) **Surgery** → (Therapeutic keratoplasty).



# Acanthamoeba keratitis (Protozal Keratitis)

**Etiology:** It is a free living organism in fresh water.

**Specific predisposing factors are:**

**1) Contact lens wearers:**

- a) Improper cleaning using home made solution.
- b) Cleaning using saliva.

**2) Traumatic abrasion.**

**Clinical picture:**

**Symptoms:** Severe pain.

**Signs:**

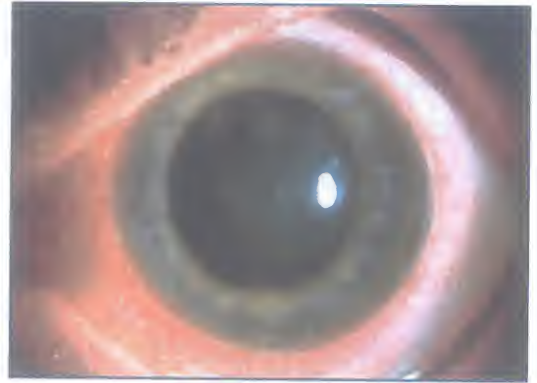
- ❖ Lesion takes the form of punctuate erosions and broad dendrites.
- ❖ Stromal crescent or annular infiltration with satellites and single or double immune ring.
- ❖ Thickened stromal nerves.

**Investigations:** As fungal keratitis.

**Treatment:**

- ❖ Diamidines (brolene).
- ❖ Neomycin.
- ❖ Ketoconazole.
- ❖ Chlorohexiden (very irritant)

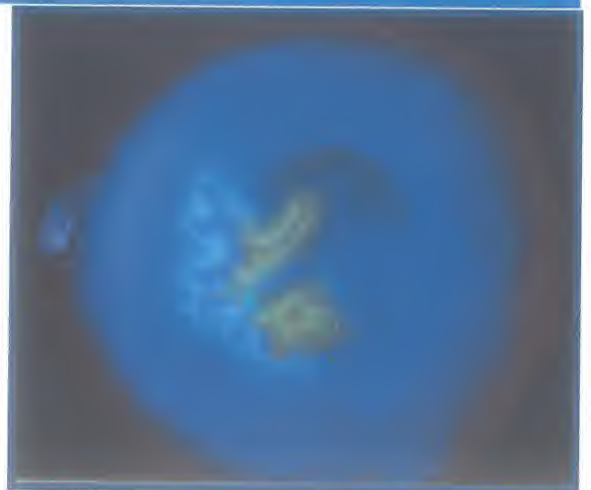
**Surgical treatment:** Therapeutic keratoplasty.



# Herpes simplex keratitis

**Herpes simplex virus (HSV):**

- ❖ Humans are the only host.
- ❖ Epithelio-tropic virus.
- ❖ Two types:
  - ✓ **HSV type 1:** cause infection above waist.
  - ✓ **HSV type 2:** cause infection below waist.
  - ✓ **HS keratitis** caused mainly by **type 1** and rarely caused by type 2.  
(Neonates during passage through birth canal).



## 1) Primary HSV infection:

❖ Occurs around age of 6 months to 5 years.

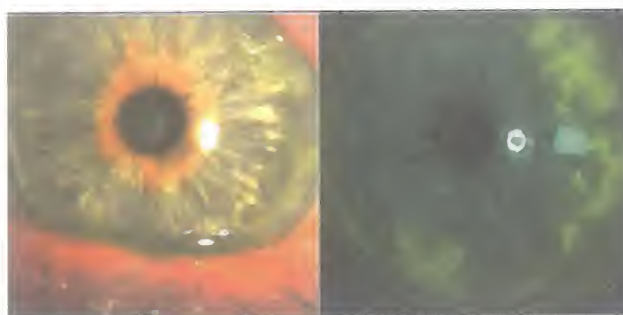
❖ Superficial lesions (stroma free).

a) **Punctuate epithelial erosions (P.E.):** Greyish white spots stained with fluorescein (epithelial defects).

b) **Punctuate Keratitis:** Granular opalescent cells stained with Rose Bengal stain. (Cells infected by virus and shows inclusion bodies).

c) **Lid vesicles.**

d) **Conjunctival Follicles.**



## 2) Recurrent HSV keratitis:

### Explanation:

❖ Following primary infections, the virus travels up along the axons of the sensory nerves to reach the sensory root ganglion.

(HSV1 Trigeminal root ganglion) ----- (HSV2 Spinal ganglion).

❖ In some patients latent stage become reversed the dormant virus become reactivated and travels down the axons to its target tissue.

### Features:

a) Typical dendritic ulcer:

- ❖ Recurrent.
- ❖ Linear, branching ulcer (dendritic).
- ❖ Superficial (Never vascularize and Never perforate).
- ❖ Ending in Knobs (cells infected e virus at the edge of the ulcer).
- ❖ Stained by double stain fluorescein for the bed of the ulcer and Rose Bengal for the knobs.
- ❖ Hypothesia.



b) Atypical or non classical forms:

❖ **Geographic or amoeboid ulcer:** → (large epith defect): Due to use of topical steroids.

❖ **Deepening of the ulcer:** Due to 2<sup>nd</sup> bacterial infection occurs Or use of steroids.

❖ **Limbal ulcer.**

❖ **Disciform Keratitis** (deep stromal lesion) Antibody reaction it may resolve without scar.

❖ **Deep necrotizing keratitis:** Actual stromal invasion by the virus itself (dense scarring and heavy vascularization).

❖ **Anterior uveitis and trabeculitis.**





## **Treatment:**

1) **Anti-viral drugs:** Interfere with viral DNA synthesis which is important for virus replication but also interfere with DNA synthesis of epithelial cells (to be stopped after 2 weeks) except acyclovir. Inhibit DNA synthesis in the virus only???

### **a) Topical:**

- ❖ **Acycloguanosine (3%) oint. (Zovirax) or acyclovir** 5 times daily.
- ❖ **Vidarabine (3%) oint.** 5 times daily.
- ❖ **Trifluoro-thymidin (T3F) (1% drops)** 5 times daily or every 2 hours.
- ❖ **IDU (5 iodo - 2 deoxy-uridine) 0.1 % drops – 0.5 oint.** rarely use toxicity.

### **b) Systemic Antiviral:**

- ❖ **(Systemic acyclovir) 800 mg tablets** 3 times daily for 3 weeks.
- ❖ **(Valacyclovir)** pro-drug rapidly converted to the Active drug acyclovir, producing greater serum concentration of acyclovir with small oral dosing.

## **2) Debridement:**

a) **Effective way** in ttt of dendritic especially if combined with antiviral.

b) **Method:** corneal surface is wiped with a sterile cotton-tipped bud 2 mm beyond the edge of the ulcer because the pathology extends beyond the dendrite

→ Rupture cells infected with the virus & protect adjacent healthy cell.

### **c) Indication:**

- ❖ Patient non compliant for antiviral.
- ❖ Allergy to antiviral.
- ❖ If antiviral not available.

d) **Contra indications:** Amoeboid ulcer + (Usual ttt: Antibiotic E.D. + Atropine + Patching).

## **Trophic ulcer**

❖ May follow HSV keratitis.

## **Causes:**

- 1) Loss of corneal sensation.
- 2) Destruction of epithelial anchoring mechanisms.

## **Treatment:**

- 1) Surface lubricants.
- 2) Bandage contact lens.
- 3) Tarsorrhaphy.
- 4) Conjunctival flaps.
- 5) Limbal stem cell grafts.
- 6) Lamellar keratoplasty.



# Herpes zoster ophthalmicus

**Introduction:** Human Herpes virus 3 (HHV-3) causes two different conditions: chicken pox (Varicella) and herpes zoster.

→ Herpes Zoster commonly affects elderly and rarely children.

## **Explanation:**

- ✚ Attack of chicken pox → Some cases the virus is retained within the sensory root ganglion
- ✚ The virus may return again along sensory nerves under influence of certain trigger factors.
- ✚ 15% of all cases of Zoster the virus affects the ophthalmic division of trigeminal (HZO).
- ✚ Involvement of Naso-ciliary branch of ophthalmic division leads to affection of the eye and the tip of the nose (Hutchinson's sign).

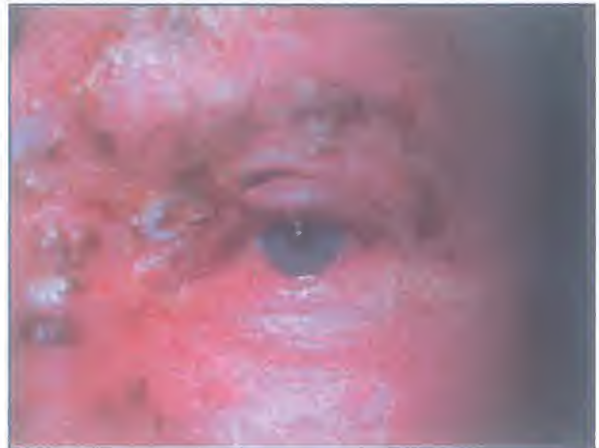
## **Clinical picture:**

### Prodroma:

- ❖ Fever.
- ❖ Headache.
- ❖ Malaise.
- ❖ Neuralgia along distribution of the nerve.

### Skin lesions:

- ❖ **Along one or more of the three branches:** (Frontal - Lacrimal - Naso-ciliary).
- ❖ Macule (red area) → papule → pustule → crusting ulcer → **healing by punched out dark scar.**



### Ocular lesions:

- ❖ **Conjunctiva:** Muco-purulent conjunctivitis.
- ❖ **Sclera:** Scleritis & Episcleritis.
- ❖ **Cornea:** (Punctate keratitis – micro-dendritis – Disciform Keratitis – nummular keratitis).
- ❖ **Uveal tract:** Anterior uveitis.
- ❖ **Retina:** Acute retinal necrosis

### Neurological:

- ❖ **Cranial nerve affection:** 2<sup>nd</sup> - 3<sup>rd</sup> - 4<sup>th</sup> - 6<sup>th</sup> (most common 3<sup>rd</sup>)
- ❖ Encephalitis.

## **Treatment:**

**1) Systemic acyclovir (tab 800 mg) 5 times daily for one week.**

*N.B. Both Famciclovir and Valacyclovir (500mg) 3 times daily*

*have the same effect as acyclovir*

**2) Skin lesions: Antiviral skin cream & Steroids skin cream.**

### 3) Ocular lesions:

- a) Blepharitis & conjunctivitis: **Cool compresses, topical lubrication and topical antibiotics** for 2<sup>ry</sup> infections.
- b) Stromal keratitis: **Topical steroids.**
- c) Neurotrophic keratitis: **Topical lubrication, topical antibiotics** for 2<sup>ry</sup> infections and **tissue adhesives and protective contact lenses** to prevent corneal perforation.
- d) Uvitis: **Topical steroids, oral steroids, oral acyclovir and cycloplegics.**
- e) Scleritis & epi-scleritis: **Topical non-steroidal anti-inflammatory.**
- f) Acute retinal necrosis: **Intravenous acyclovir (1500 mg per day divided into 3 doses) for 7-10 days.**

## Non infective corneal ulcer

- 1) Ulcer associated with systemic collagen vascular disorders.
- 2) Traumatic corneal ulcer.
- 3) Neurologic Keratitis.
- 4) Kerato-malacia.
- 5) Mooren's ulcer.
- 6) Phlyctenular ulcer

## 1- Neurologic Keratitis

**Definition:** Corneal ulcer due to nerve lesions whether sensory or motor and so it is classified into:

- 1) **Motor type** (ulcer with lagophthalmos).
- 2) **Sensory type** (Neuro-trophic keratitis).

### Ulcer with lagophthalmos: (Exposure Keratopathy).

**Definition:** Ulcer associated with facial nerve paralysis leading to orbicularis paralysis and lagophthalmos.

#### Explanation:

- ❖ Lack of corneal protection from minor trauma.
- ❖ Dryness of the cornea necrosis and sloughing of superficial corneal layers.

**Clinical picture:** Horizontal oval ulcer involving lower third of the cornea (why??).

**N.B** Ulcer may be central (why??)

#### Treatment:

##### 1) Prophylaxis against ulcer:

- a) Topical lubricants (artificial tears E.D) at day time and topical ointment at night.
- b) Closure of the lids by adhesive tape during sleep.
- c) Lateral tarsorrhaphy.
- d) Therapeutic contact lenses.

##### 2) Treatment of the ulcer if it forms. (As before).



## Corneal anesthesia: (Neurotrophic Keratitis).

**Definition:** Corneal ulcer due destruction of trigeminal ganglion of nerve,  
**which may be due to:**

- 1) **Traumatic:** Fracture base of Skull.
- 2) **Congenital: Riley - Day syndrome:**
  - a) Corneal anesthesia.
  - b) Corneal dryness.
  - c) Congenital insensitivity to pain.
- 3) **Inflammatory:** Post Herpes simplex & Zoster – Gummatous meningitis.

### **Causes:**

- 1) **Congenital:** e.g.: Familial dysautonomia (Riley-Day syndrome): Corneal anesthesia, epithelial erosions, dry eye and congenital sensitivity to pain.
- 2) **Inflammatory:** Herpes simplex, Herpes zoster and Leprosy.
- 3) **Traumatic:** Facial or skull trauma.
- 4) **Neoplastic:** Acoustic neuroma.
- 5) **Vascular:** Aneurysms.
- 6) **Iatrogenic:** Contact lens wear, trauma to ciliary nerves and corneal incisions.
- 7) **Local diseases:** Corneal dystrophies as Lattice and Granular.
- 8) **Systemic diseases:** DM, Vit.A deficiency and multiple sclerosis.
- 9) **Miscellaneous:** Increasing age as well as loss of trophic nerve impulses.
- 10) **Topical medications:** Anesthetics, Timolol, Sulfacetamide, Diclofenac

**Explanation:** Loss of corneal sensation loss of reflex blinking and lacrimation.

Mechanism: Production of harmful antidromic impulses.

### **Clinical picture:**

Central C.U e rapid progression up to perforation.

### **Treatment:**

- 1) **If recovery of sensation is expected:**
  - a) Topical lubricants.
  - b) Bandage C.L.
  - c) Temporary tarsorrhaphy.
- 2) **If recovery will not likely to occur:**
  - a) Conjunctival flap.
  - b) Permanent tarsorrhaphy.
  - c) Botulinum toxin injection may be used instead of tarsorrhaphy to induce temporary ptosis and protect the globe,
  - d) Therapeutic keratoplasty (if perforation is impending).

## 2- Keratomalacia

**Definition:** Acute corneal melting due to vitamin A. deficiency.

**Etiology:** Advanced starvation and marasmus in young children.

**Clinical picture:**

- ❖ Loss of corneal and conjunctival luster (unhealthy epith.).
- ❖ Bilateral corneal melting overnight.
- ❖ Prolapse of intraocular contents.
- ❖ 2<sup>nd</sup> bacterial infections.

**Treatment:**

- ❖ Large doses of Vit .A.
- ❖ Improve general health.
- ❖ Routine treatment of C.U.



## 3- Photophthalmia

**Definition:** Superficial keratitis caused by U.V. rays (snow blindness, exposure to an ultraviolet lamp, arc welding and high tension electric current).

**Clinical picture:**

**Symptoms:** Pain – Blepharospasm – Photophobia – Lácimation.

**Signs:**

- ❖ Latent period of 4-5 hours between exposure and ...?
- ❖ Multiple superficial corneal erosion.

**Protection:** Dark glasses and special protective goggles that absorb UV rays Should be worn when exposure to hazardous rays is anticipated.

**Treatment:**

- ❖ Lubricants
- ❖ Bilateral patching for one day.
- ❖ Cold compresses.

## 4- Marginal corneal ulcer

### a) C.U associated with systemic collagen vascular disorders

**Definition:** C.U. associated with auto immune diseases such as S.L.E., rheumatoid arthritis, poly-arthritis nodosa and wagner's granulomatosis

**Etiology:**

- ❖ Vasculitis involving limbal capillaries causing its obliteration, which when occurs indicating wide spread vasculitis have occurred,
- ❖ Dryness associated with these diseases also an additional factor.

**Clinical picture:** Marginal corneal ulcer.

**Treatment:**

- ❖ Lubricants.
- ❖ Cycloplegic E.D.
- ❖ Systemic Immunosuppressive therapy

### b) MOOREN'S ULCER

**Definition:** Chronic slowly progressive peripheral corneal ulceration which has a serpiginous pattern.

**Etiology:** May be:

- ❖ Immunological cause which leads to obstructive Vasculitis of limbal capillaries.
- ❖ The adjacent conj. produces destructive enzymes (collagenase and protoglycans).

**Clinical picture:** There are two clinical types:

- 1) **Unilateral type:** (Benign – Less severe – Affects elderly patients).
- 2) **Bilateral type:** In younger patients.

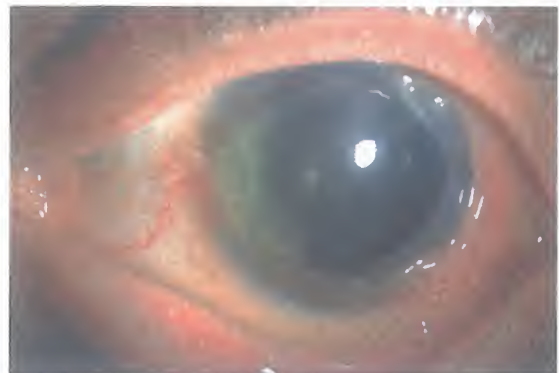
**N.B:** The ulcer is also known as chronic serpiginous ulcer

**N.B.2:** There are 3 serpiginous ulcers:

- 1) Typical hypopyon ulcer.
- 2) Mooren's ulcer.
- 3) Phlyctenular (Fascicular ulcer).

**Treatment:**

- 1) Immuno-suppressive therapy.
- 2) Excision of peri-limbal conjunctiva.
- 3) Therapeutic keratoplasty.





## c) Phlyctenular Ulcer

(See conjunctiva)

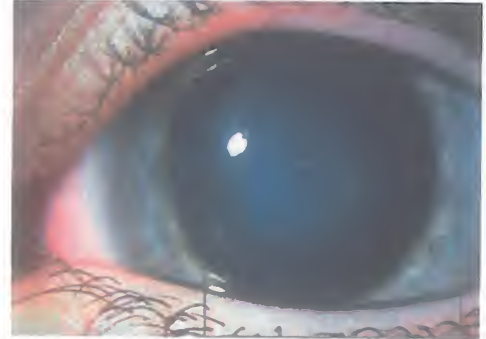
## Interstitial Keratitis

**Definition:** It is a non-suppurative infiltration affecting stroma

**Morphological Classification:**

**1) Diffuse or sectorial keratitis:**

- a) Syphilis (Congenital, or acquired).
- b) TB.
- c) Leprosy.
- d) Viral infection (HSV & HZV).



**2) Disciform keratitis:** Herpes simplex (commonest cause).

**3) Nummular keratitis:** Multifocal round infiltrations (herpes Zoster).

### Interstitial keratitis: (Due to syphilis)

- ❖ 90% of all cases of I.K. is due to Syphilis.
- ❖ 87% of cases due to cong. Syphilis.
- ❖ 3% of cases due to acquired Syphilis.

**Etiology:** Ag-ab reaction to *Treponema Pallidum* Toxius is the cause of I.K.

**Clinical picture:**

❖ **Age:** Between 5 - 25 years.

❖ **Side:** Bilateral.

❖ **Signs:**

- 1) Progressive stage (2 weeks): Infiltration & vascularization of stroma.
- 2) Florid Stage (2 months): Extensive infiltration and vascularization (Salmon patch appearance).
- 3) Regressive stage (2 years): Clearing of infiltration but vascularization remains (ghost vessels).

### N.B.: Hutchinson's triad of cong. Syphilis.



### **Treatment:**

- 1) Topical & S cortisone E.D.
- 2) Topical Cycloplegic E.D.
- 3) Systemic penicillin.
- 4) Kerato-plasty.

## Corneal Dystrophies

**Definition:** Group of corneal affections characterized by:

- 1) Usually inherited.
- 2) No inflammatory signs.
- 3) Bilateral.
- 4) Stationary or slowly progressive.
- 5) Affects adults.

### **Classification:**

- 1) Anterior dystrophies: Involving the epithelium, e.g.: recurrent corneal erosion syndrome.
  - ❖ **Erosion** is caused by cornea scratches with --- e.g. finger nails
  - ❖ **Recurrence** occurs with any slight trauma due to imperfect healing of basement membrane (AMBD)
- 2) Stromal dystrophies: Involving the stroma, e.g.: Lattice dystrophy.
- 3) Posterior dystrophies: Involving the endothelium, e.g.: Fuch's endothelial dystrophy.
- 4) Ectatic dystrophies: Keratoconus.

## Recurrent corneal erosion syndrome

RCES may be traumatic, infective, or degenerative. The condition starts by a corneal abrasion that heals in a few days. However, shortly later spontaneous recurrence of the abrasion happens in the same site of the original pathology, usually on waking up in the morning.

### **Pathophysiology:**

Recurrent corneal erosions occur because there is a **defect in the epithelial basement membrane adherence and in hemidesmosomes formation between epithelial cells, resulting in epithelial loss, microcysts, and bullae**. On waking up in the morning the upper lid peels off the weakly adherent epithelium.

**Symptoms:** Mild to severe eye pain is the primary symptom.

### Other symptoms:

- ❖ Slightly blurred vision (when the epithelial and basement, changes are in the visual axis)
- ❖ Visual acuity loss
- ❖ Epithelial blebs
- ❖ Foreign body sensation with recurrent erosion, when the epithelium loosens,

### **Signs:**

Depending on the severity of the erosion, corneal examination findings may be totally normal, or they may reveal signs of RCE syndrome.

- ❖ During an acute attack, one may see **epithelial loss, epithelial microcysts, bullae, lack of adherence of sheets of epithelium, and epithelial filament formation**. In these instances, the visual acuity may be impaired severely if the pathogenic condition occurs in the pupillary area.

### **Lines of treatment:**

In mild cases, the condition may resolve spontaneously within a few hours. However, more often, treatment is required to promote healing and to relieve the symptoms.

- ❖ If the erosion is small, it usually heals spontaneously or with the aid of the following:
  - **A pressure patch** placed on the eye for 1 - 2 days
  - **An antibiotic ointment**, which can be used beneath the patch. This also acts as a lubricant.
  - Sometimes these measures must be followed for **several months after resolution of the episode**.
- ❖ In some cases of multiple recurrent erosions, soft contact lenses can be helpful. Bandage lens treatment, if used for this indication must be continued for up to 8-26 weeks to facilitate repair of the corneal epithelial basement membrane.
- ❖ However, **persistent use of soft contact lens** increases the risk of infectious corneal disease.
- ❖ Resistant cases may require the following:
  - **Mechanical debridement, with or without chemical cautery**, depending on the size of the defect and the amount of ocular irritation
  - **Local cycloplegic agents**
  - **A diamond burr**, which is used to "polish" the Bowman layer after mechanical debridement (proven to be effective in preventing recurrences).
- ❖ Delaminating the corneal epithelium using alcohol 20% can improve the symptoms of recurrent corneal erosions in eyes that do not respond to topical lubrication or bandage contact lenses.
- ❖ Surgical procedures: such as **Anterior stromal puncture, Excimer laser, phototherapeutic keratectomy (PTK)** and may reach up to corneal grafting.



# Keratoconus

**Definition:** It is a progressive central and para-central stromal thinning leading to corneal ectasia and apical protrusion.

**Characters:**

- 1) **Bilateral (85%).**
- 2) **Asymmetrical.**
- 3) **Start around puberty ( 10 - 20 years).**
- 4) **Progress for few years (variable).**
- 5) **Common associations are:**
  - a) Systemic diseases:
    - ❖ Down's syndrome.
    - ❖ Marfan's syndrome.
  - b) Local eye diseases:
    - ❖ Spring catarrh.
    - ❖ Retinitis Pigmentosa
    - ❖ Contact lens wearers may be met with in cases of keratoconus.



**Clinical picture:**

**Symptoms:** Rapid progressive diminution of V.A and frequent changes of glasses.

**Explanation:** Progressive Myopia and astigmatism

**Signs:**

## Mild keratoconus

- 1) **External and corneal signs:** are absent or minimal.
- 2) **A history of:** multiple inadequate spectacle corrections of one or both eyes may be noted and may include oblique astigmatism on refraction as well as moderate to high myopia.
- 3) **Keratometry:** Irregular astigmatic Keratometry values
- 4) **Placido disc:** Irregular circles with central crowding.
- 5) **Corneal topography:** Diagnosis can be confirmed.

## Moderate keratoconus

1) **Gross sign:** Munson sign (on looking downwards causing angulation of lower eyelids).

2) **Slit lamp signs:**

- a) Enhanced appearance of corneal nerves is noted.
- b) Vogt striae (fine stress line): in the deep stroma.
- c) Corneal scarring (superficial or deep).
- d) Deposition of iron in the basal epithelial cells in a ring shape at the base of the conical protrusion called the Fleischer ring.



3) **Keratometry:** Typically increase to **45-52 D**.

4) **Retinoscopy:** Distortion of red pupillary reflex may allow observation of "scissoring" or an inferior distortion termed the oil drop sign.

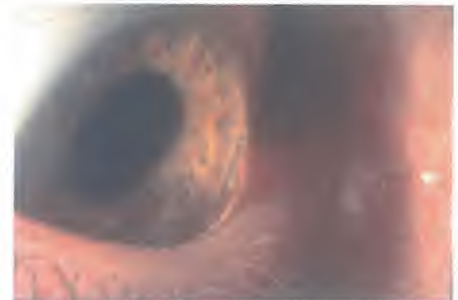
## Advanced keratoconus

1) **Slit lamp:** Vogt striae in 60% of eyes and Fleischer ring and/or scarring in 70% of eyes.

2) **Keratometry:** **Greater than 52 D** and enhancement of all corneal signs, symptoms and visual loss/distortion.

3) **Complication: Acute corneal hydrops:**

- ❖ Continuous stretch and ectasia of the cornea leads to break in DM → acute corneal edema and opacification,
- ❖ Resolves in few weeks leaving a residual central opacity.



## **Management:**

1) **Spectacles** can be used in early cases before astigmatism becomes irregular.

2) **Rigid C.L.** may help in irregular astigmatism but are useless in case of opacity or steep cones.

3) **Cross linking**

4) **Surgical procedures:**

- a) **Intra-stromal corneal rings:** For patients who become intolerant to contact lenses. More successful in modest than advanced disease.
- b) **Lamellar keratoplasty:** especially deep anterior lamellar keratoplasty (DLAK).
- c) **Corneal transplants (penetrating keratoplasty PKP):** very successful in Keratoconus resulting in clear visual axes in more than 90% of cases.

# Corneal degenerations

## 1 - Arcus senilis

**Definition:** It is annular infiltration of the corneal periphery by lipid material which is seen in old aged.

**Clinical picture:**

**Symptoms:** (doesn't affect vision).

**Signs:** White ring (1-2 mm broad) separated from the limbus by a clear zone lucid interval of Vogt

**D.D.:** From pannus by the presence of lucid interval of Vogt.

**Treatment:** (NO treatment).

**Rule of 2:**

- ❖ Upper and lower arcs that meet to form a ring.
- ❖ Superficial and deep triangular stromal infiltration with apices meeting.
- ❖ Corneal and scleral infiltration with clear interval.

N.B.: Arcus senilis is an aging process, but if seen in young → Arcus juvenilis  
(which may indicate lipemia & hypercholesterolemia)



## 2 - Band Keratopathy

- ❖ A horizontal faint band shaped corneal opacity.
- ❖ It is due to  $\text{Ca}^{++}$  deposition at the level of Bowman's membrane.
- ❖ It shows rounded holes where nerves pass.

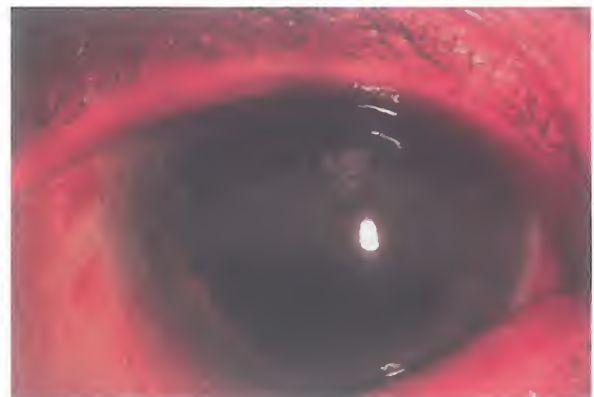
**Etiology:**

- 1) Chronic anterior uveitis (young age).
- 2) Idiopathic age related (elderly).
- 3) Phthisis bulbi.
- 4) Elevated serum  $\text{Ca}^{++}$  or P.

**Clinical picture:**

**Symptoms:**

- ❖ Decreased vision.
- ❖ Foreign body sensation,
- ❖ Ocular sensation.
- ❖ Redness.



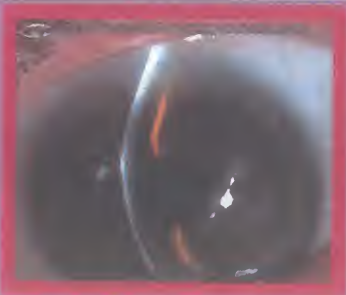



### Signs:

- ❖ **Visual acuity:** Will be decreased in proportion with the density of calcium deposits
- ❖ **Slit lamp:** Whitish-grayish plaque like deposition that occurs in a **band across the cornea**.  
Holes in the **plaque** may be apparent, representing spaces where the corneal nerves are traversing the Bowman membrane to the epithelial surface.

### Treatment:

- 1) Scraping and washing the cornea with **Ethylene Diamine (EDTA) Tetra- acetate solution**.
- 2) Treat the cause.

CORNEAL OPACITIES		
	1) Nebula	2) Leucoma
Peripheral	Glasses or hard C.L.	Glasses or hard C.L.
Central	<ul style="list-style-type: none"><li>❖ Photorefractive Keratectomy (Eximer Laser).</li><li>❖ Lamellar Keratoplasty</li></ul>	<ul style="list-style-type: none"><li>❖ Penetrating Keratoplasty</li><li>❖ Visual Iridectomy</li></ul>
		

### 3) Leucoma adherent:

- 1) **No increase (I.O.P.):** manage as leucoma non adherent.
- 2) **Increase (I.O.P.) :**
  - a) **Seeing eye:** lower I.O.P. by external fistulizing surgery then manage as leucoma non- adherent:
  - b) **Non-seeing eye:** lower I.O.P. to relieve pain then use colored C.L. (black pupil) or cosmetic keratoplasty or tattooing.

### 4) Anterior staphyloma:

- 1) **Partial anterior staphyloma:**
  - a) **Seeing eye:** (Glaucoma surgery – Synechiolysis then keratoplasty).
  - b) **Non-seeing eye:** Enucleation.
- 2) **Total anterior staphyloma:** Enucleation.

## Management of corneal opacities depends on several factors:

1) **Retinal and optic nerve functions should be normal.** Assessment of retinal and optic nerve functions in an eye with opaque media runs along the same line as in eyes with mature cataract and includes ultrasonography, macular function tests and peripheral retinal function tests. Electrophysiological tests (ERG, VEP) may be needed

2) **The intra-ocular pressure should be first controlled if high**

## Following this, and according to the site, size and density of the opacity different lines of treatment are available:

- 1) **For a central loucoma, penetrating keratoplasty** is the best treatment nowadays. A few decades ago, a **visual iridectomy** might have been a reasonable alternative.
- 2) **Peripheral scars** are managed according to the astigmatism they induce. A peripheral scar may be **left alone, corrected with glasses, with rigid CL or surgically with release incisions**. If the only problem is cosmetic, then a **colored CL** can solve the problem.
- 3) **Nebulae causing irregular astigmatism** are managed with **rigid CL, lamellar keratoplasty or excimer laser (photo-therapeutic keratectomy: PTK)**.
- 4) **Older techniques** such as **visual iridectomy and tattooing of the cornea** have little place in modern ophthalmic practice.

## KERATOPLASTY

The replacement of diseased corneal tissues. The cornea may be obtained from the other eye of the same patient (autogenous graft), or from cadaver eyes (allograft).

### **1) Autogenous grafts:**

The donor may be the other eye of the same individual as in cases of where one eye has an **opaque cornea** with **normal retina and optic nerve**.

→ This serves as **the recipient eye**.

The other eye has a **clear cornea** but a **diseased retina and nerve**.

→ This serves as **the donor eye**.

### **2) Allograft:**

Taken from cadavers of the same species (human cadavers) are the standard technique used.

✚ The cornea is obtained a few hours after death, and preserved in a special medium to keep the endothelium viable for few days until the keratoplasty is performed.

✚ Corneas obtained in this way are kept in "Eve - banks" where the donor tissue is examined to make sure that its endothelium is suitable and that the tissue is free from communicable diseases such as AIDS, HBV, HCV, rabies and other viral and bacterial pathogens.

✚ Corneal grafting may be penetrating (full thickness graft) or lamellar (partial thickness graft).

- ✚ Corneal transplanting has high rate of success because the cornea is an organ with immunological privilege. This is because the healthy cornea is avascular, free of lymphatics and its cellular population is comparatively low. These factors render graft rejection less common than any other transplant in the body.

#### **Indications of keratoplasty include:**

- 1) Corneal opacities.
- 2) Impending and actual corneal perforation.
- 3) Keratoconus.
- 4) Corneal fistula.
- 5) Corneal ulcers not responding to medical treatment such as some fungal and protozoal ulcers



#### **Complications:**

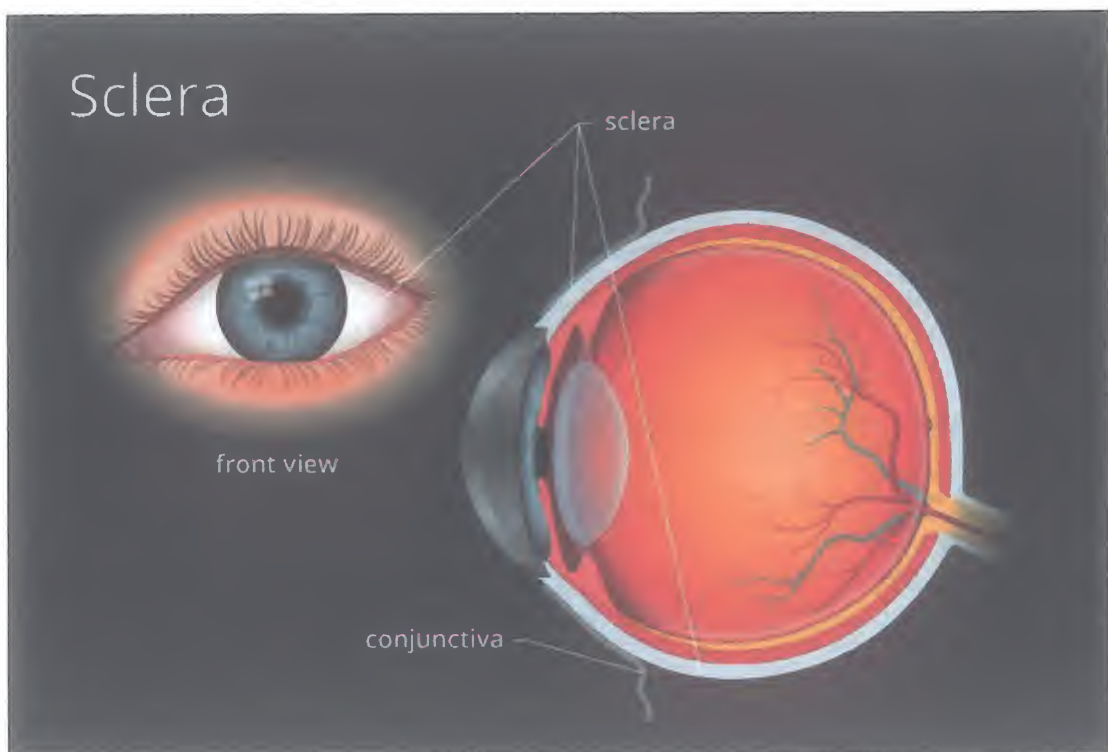
- ✚ **Early complications:** Flat anterior chamber, persistent epithelial defects and infection.
- ✚ **Late complications:** Glaucoma, astigmatism, late wound separation, cystoid macular edema and recurrence of the initial disease in the donor graft.
- ✚ **Graft failure:**
  - ❖ **Early:** Cloudiness of the cornea from the first post-operative day.  
**Caused by:** Defective donor endothelium or trauma during surgery.
  - ❖ **Late:** Usually the result of immune graft rejection. 50% occur in the first 6 months and the majority occurs in the first year post-operative.

#### **Treatment:**

Hourly topical steroids as well as peri-ocular steroid injection and/or oral steroid depending on the clinical picture.



# Sclera



# Sclera

## Anatomy

❖ It is the posterior  $\frac{5}{6}$  of the outer coat.

❖ It is formed of:

- 1) Tenon's capsule.
- 2) Epi-sclera.
- 3) Lamina fusca

❖ Perforated by:

- 1) **Arteries:** Ant. ciliary a. & post, ciliary a
- 2) **Veins:** Ant. ciliary v. & vortex veins.
- 3) **Nerves:** Short & long ciliary nerves.

## BLUE SCLERA

### Causes:

1) Physiological: as in infants & children.

2) Pathological:

- a) Buphthalmos.
- b) High myopia.
- c) Over staphyloma.
- d) Osteogenesis imperfecta: Blue sclera, deafness & fragile bone.

→ In all these cases, the sclera is thin showing the underlying uvea (choroidal vessels).

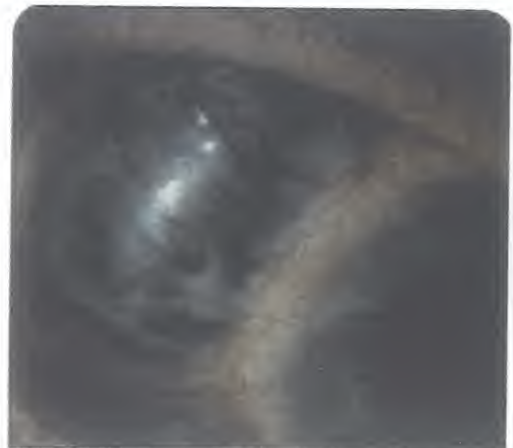


## Staphyloma

**Definition:** It is bulging of the outer coat of the eye (cornea or sclera) which is lined by the uveal tissue.

**Etiology:** (1+ 2) together

- 1) Weak outer coat of the eye due to ocular disease:
  - a) Corneal diseases e.g. perforated corneal ulcer.
  - b) Scleral diseases e.g. high axial myopia.
- 2) ↑ IOP e.g. absolute glaucoma.



## Types

**1) Anterior Staphyloma:** ectatic corneal scar + adherent iris

(Anterior staphyloma = corneal staphyloma = partial or total).

### **Etiology:**

- 1) Large corneal perforation → **iris prolapse**.
- 2) The iris soon covered by fibrin → **fibrous tissue**.
- 3) The weak scar (large defect) cannot withstand the (↑ IOP due to closure of the angle by the iris) → **ectasia**.

V.P. In total staphyloma the whole cornea is detached & perforated.

Staphylomatous tissue = pseudo-cornea

**D.D...:** From Keratectasia (see cornea).

**Treatment:** See the cornea.

**2) Scleral:** ectatic sclera lined by uveal tissue.

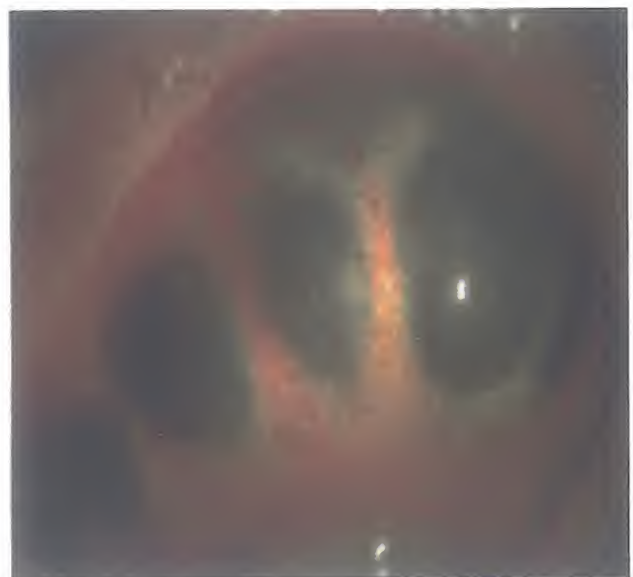
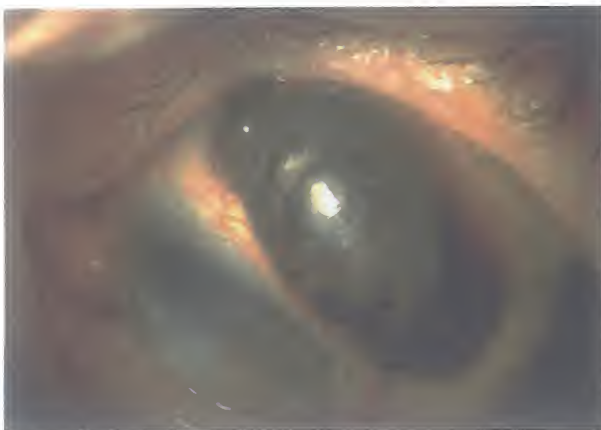
### **Causes:**

- ❖ Scleritis
- ❖ Absolute glaucoma,
- ❖ Trauma: rupture
- ❖ Progressive myopia.

### **Types:**

- 1) **Equatorial:** Lined by choroid
- 2) **Ciliary:** Lined by ciliary body
- 3) **Intercalary:** Lined by the root of the iris
- 4) **Posterior:** Occurs at the posterior pole of the eye. It occurs only in high myopia

**Treatment:** Treatment of the cause.





# Episcleritis

✚ It is inflammation of the epi-scleral tissue. It is more common in females.

## **Etiology:**

- 1) **Idiopathic:** most common.
- 2) **Allergic reaction** to an endogenous toxin as a septic or tuberculous focus.
- 3) **Collagen disease** as in rheumatoid arthritis.

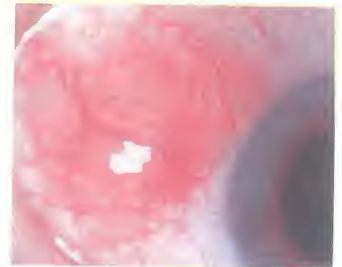
## **Symptoms:**

- 1) Discomfort and lacrimation.
- 2) Tenderness on pressing the globe.

## **Signs:**

### 1) **Nodular type:**

- a) Lentil size, well circumscribed.
- b) **Purple in color** due to dilatation of the deep vessels.
- c) It is tender and the conjunctiva moves over it????



### 2) **Diffuse type:**

- a) Dilated epi-scleral vessels.
- b) Frequently affecting only one sector.
- c) No nodule.



**Differential diagnosis:** Episcleritis should be differentiated from phlycten.

## **Treatment:**

- 1) Topical steroids.
- 2) Phenyl-butazone orally.

# Scleritis

✚ In scleritis Inflammation affects all layers of the sclera.

✚ It is more serious than epi-scleritis.

## **Etiology:**

- 1) 50% of cases are associated with rheumatoid arthritis.
- 2) 50% of cases are idiopathic.

## **Symptoms:**

- 1) Severe pain.
- 2) Photophobia and lacrimation.



## **Signs:**

### **1) Anterior type:**

- a) **Nodular:** the nodule is deep and fixed to the sclera.
- b) **Diffuse.**
- c) **Annular.**
- d) **Necrotizing:** serious and may result in perforation of the globe.
- e) **Scleromalacia perforans:** melting of the sclera in a quiet eye.

### **2) Posterior type:** Causes severe pain and uveitis with exudative retinal detachment.

## **Complications:**

### **1) Uveitis.**

### **2) Sclerosing keratitis:** Opacity develops in the cornea near the scleral nodule.

### **3) Scleral staphyloma:** Due to weakness of sclera.

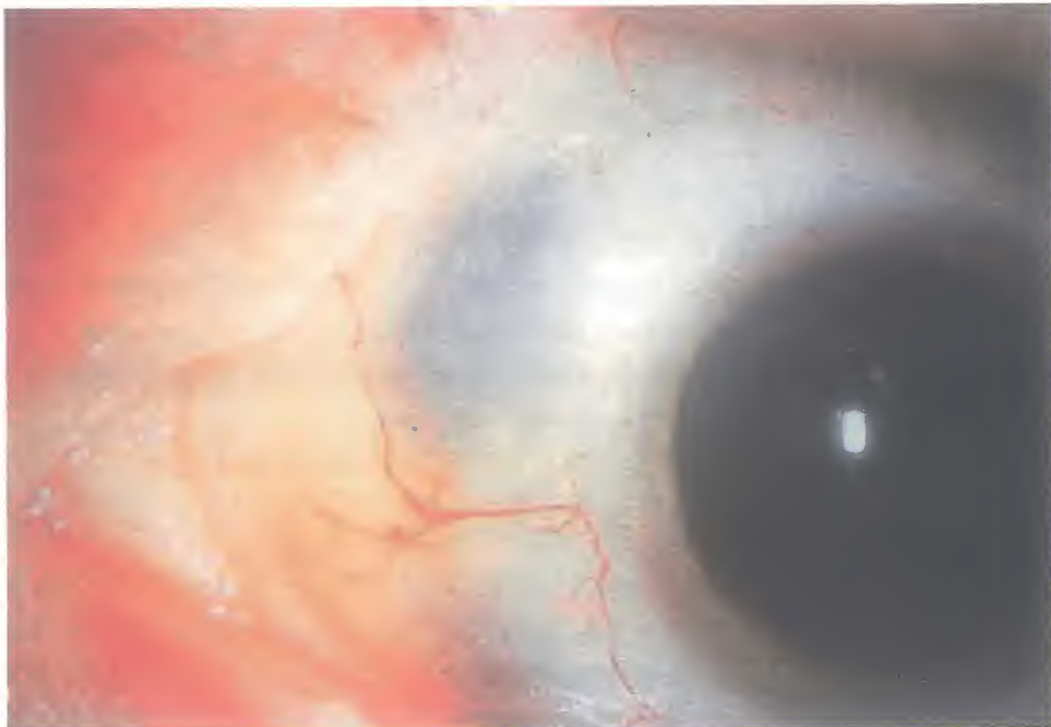
## **Treatment:**

### **1) Topical steroids.**

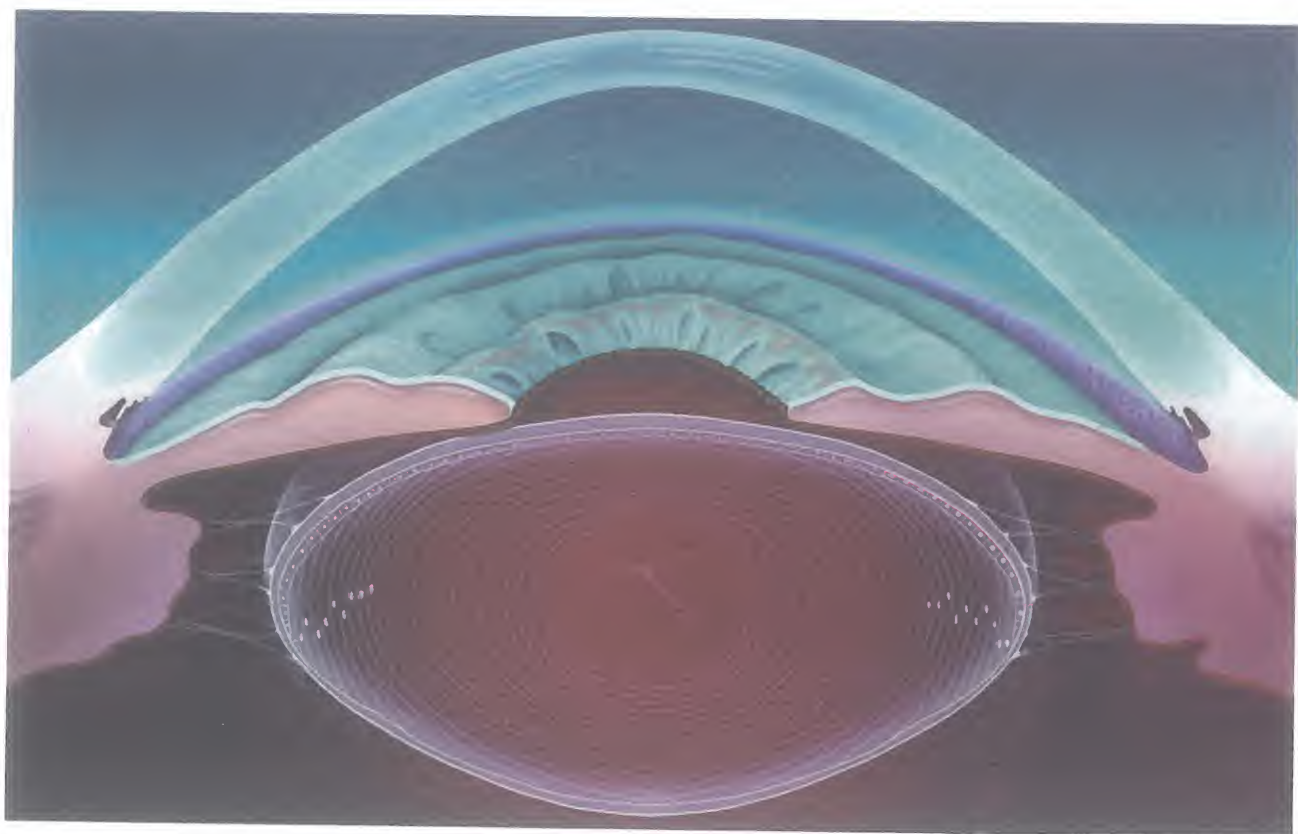
### **2) Oral phenyl-butazone.**

### **3) Large dose of oral steroids.**

### **4) Immuno-suppressives.**



# The Crystalline Lens





# The Crystalline Lens

## Anatomy

It is an:

- ❖ Elastic.
- ❖ Transparent.
- ❖ Avascular.
- ❖ Biconvex structure which is suspended in its place by the suspensory ligament (Zonules).

### Gross anatomy:

#### 1) Relations:

- ❖ **Anteriorly:** Iris (separated by Posterior Chamber),
- ❖ **Posteriorly:** Vitreous (separated by Retro-lental space).

#### 2) Shape:

##### a) surface:

- ❖ Convex (radius = 10 mm).
- ❖ Centre: is the anterior pole.

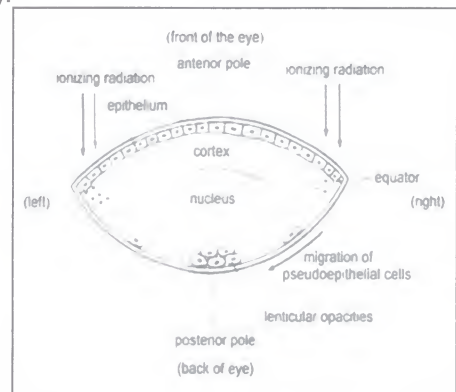
##### b) Posterior surface:

- ❖ More convex ( $r = 6$  mm).
- ❖ Center: is the posterior pole.

##### c) Axis of the lens: The line joining the ant. & post, poles (4-5 mm).

##### d) Equator (Lens edge):

- ❖ Gives attachment to the zonules.
- ❖ Eq. Diameter = 9-10 mm.



### Minute anatomy:

#### 1) Capsule:

- ❖ Highly elastic.
- ❖ Secreted by anterior sub-capsular epith
- ❖ **Thickness:** anterior capsule > post.
- ❖ **Functions:**
  - a) Accommodation.
  - b) Semi-permeable membrane (Nutrition in & waste P. out).
  - c) Protection: of lens fibers from aqueous enzymes.

#### 2) Sub-capsular epithelium:

- ❖ **Site:** Line the anterior capsule not the posterior capsule.

**N.B. Posterior capsule epithelium was changed into lens fibers during fetal life.**

- ❖ **Shape:** One layer of cubical cells that gradually elongate towards the equator.

### 3) Lens fibers:

❖ Secreted by: equatorial cells:

The new fibers are laid outside the older ones, which are pushed inward and undergo a process of sclerosis (loss of water).

❖ Divided into:

a) Peripheral fibers: soft (cortex).

b) Central fibers: hard being compressed and sclerosed (nucleus).

❖ Sutures: site of articulation of lens fibers.

(Y shaped: erect anteriorly "Y" & inverted posteriorly).

#### **N.B.: The Nucleus is divided into:**

- 1) Embryonic nucleus: 1-3 months intrauterine.
- 2) Fetal nucleus: 3-9 months (I.U.).
- 3) Infantile nucleus; up to puberty.
- 4) Adult nucleus: After puberty.

#### **Nutrition:**

❖ Before birth: Hyaloid artery.

❖ After birth: Diffusion from aqueous.

#### **Composition:**

1) Water: 65%.

2) Proteins: 34%.

3) Minerals: 1%.

#### **Metabolism:**

❖ Anaerobic: (as the lens is a vascular → no enough O<sub>2</sub>).

❖ Oxidation: occurs by dehydrogenation which requires Hydrogen carriers as Vit. C &

Glutathione

#### **Refractive index:**

❖ Cortex: 1.39.

❖ Nucleus: 1.42.

**Refractive power:** (15 – 17 D).

#### **Functions:**

- 1) One of the refractive media of the eye (converging).
- 2) Accommodation.
- 3) Protect the retina from U.V rays.

### Accommodation

**Definition:** It is the ability of the eye to change its dioptric power to see at different distances (far & near) → clear.

**Mechanism:** Contraction of ciliary ms (circular fibers) → a smaller ciliary ring + relaxation of the zonules → ↑ curvature and power of the lens.

## Cataract

**Definition:** It is lens Opacification.

### **Classification:**

#### 1) According to the cause:

- a) Congenital.
- b) Acquired (traumatic, complicated or senile).

#### 2) According to the Age:

	Soft cataract	Hard cataract
<b>Incidence</b>	Before the age of 25 years.	After the age of 25 years
<b>Includes</b>	Congenital, Traumatic & complicated cataract.	Senile, Traumatic complicated cataract.
<b>Nucleus</b>	No hard nucleus	Hard nucleus
<b>Lens Proteins</b>	(Soluble ptn.) Can be digested by aqueous enzymes, so, if the lens capsule is ruptured by trauma or operation → absorption of lens matter occurs → no after cataract.	(Insoluble ptn.) which cannot be digested by aqueous enzymes So, if the lens capsule is ruptured by trauma or operation → soft lens matter will be absorbed but the nucleus will not → after cataract.

#### 3) According to the layer affected:

- ❖ Sub-capsular.
- ❖ Cortical.
- ❖ Nuclear.

## Congenital and developmental cataract

**Definition:** It is lens opacification which dates since birth or shortly after.

### **Etiology:**

- 1) Hereditary
- 2) Malnutrition
- 3) Intra-uterine infection.
- 4) Exposure to irradiation.
- 5) Teratogenic drugs.



### Characteristics:

- 1) Commonly bilateral
- 2) Commonly associated with other congenital anomalies.
- 3) Commonly stationary course.

### Types:



## 1 - Anterior polar cataract

**Etiology:** Delayed formation of A.C. As, Prolonged contact between the cornea & lens →

Irritation of sub-capsular epithelium at the anterior Pole → Its proliferation and formation of mass of cells + Calcium deposition → Opacity.



D.D.: From acquired type (more common) due to: Small, Central corneal perforation.

	Congenital	Acquired
History of red Eyes	- ve	+ ve
Corneal Opacity	Absent	Present
Side	Bilateral	Unilateral

## 2-Posterior polar cataract

**Etiology:** Persistence of remnants of the Hyaloid artery.

**Clinically:** Disc shaped opacity at the posterior pole.

**Vision:** Markedly

(as the opacity is close to the nodal point).



## 3-Lamellar cataract (Zonular cataract)

**Definition:** It is lens opacification involving one or more lamellae of the lens.

### **Etiology:**

1) Hereditary: Malnutrition of the mother during pregnancy especially Vitamin D and  $\text{Ca}^{++}$  as the child often shows:

- a) Evidences of rickets.
- b) Abnormality of permanent teeth.

### **Clinically:**

With the pupil dilated, the lens opacities composed of:

- 1) **Central disc.**
- 2) **Projections (spokes or riders).**

This resembles: steering wheel of a ship or cart wheel appearance.

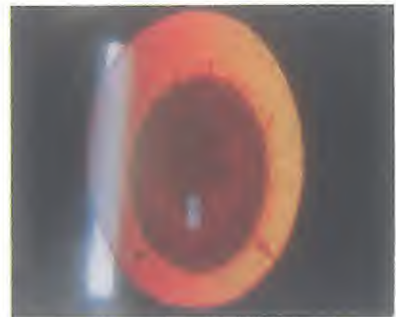
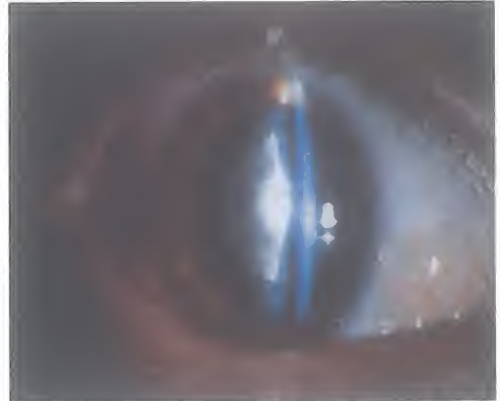
### **Explanation:**

1) **Disc:** **Vit. D & Ca deficiency** → Formation of opaque fibers.

On recovery from this deficiency, clear fibers are formed pushing the opaque fibers in ward → opaque lamella (this could be repeated).

2) **Riders:** The equatorial cells don't recover at the same time.

As some of them recover forming clear. Fibers, those beside them continue to form fibers (riders).



## 4-Total cataract

❖ Due to Rubella infection to the mother in the 1st trimester.

❖ The cataract operation is risky in this patient due to:

- a) Associated cardiac anomalies (risk during general anesthesia).
- b) The virus remains dormant inside the lens for 2-3 years (i.e. operation → endophthalmitis).

### **Other types**

- 1) **Blue dot cataract** (small dots scattered in the lens)
- 2) **Sutural cataract** (opacities at the Y sutures)
- 3) **Coronary cataract** (club shaped opacities at the equator).



## Clinical picture of congenital cataract:

**Symptoms:** Given by the mother:

- ❖ White pupil (leucoria).
- ❖ Defective vision.

**Signs:**

- ❖ One of the previous types is seen.
- ❖ Fundus should be carefully examined to exclude retinal anomalies.
- ❖ Visual assessment: Prefrential looking test (VEP)

**Complications:**

- ❖ Bilateral opacity → Nystagmous (As opacity interferes with foveal development).
- ❖ Unilateral → Amblyopia & squint

**D.D.** Other causes of leucoria (white pupil):

- |   |                       |
|---|-----------------------|
| 1) Congenital cataract.                 | 4) Retinal detachment |
| 2) Persistent hyperplastic primary vit. | 5) Retinoblastoma.    |
| 3) Retinal dysplasia.                   | 6) Coat's disease.    |

**Treatment:**

## How to asses V/A

### 1) Preverbal children:

**a) Occlusion test:** Occlusion of one eye of the child then this is the sound eye.

#### **b) Hundred (100) & thousand (1000) Sweet Test:**

- ❖ Put tiny colored sweets in your hand at a distance 33 cm (if the child moves his hands towards theses sweets then V/A is 6/24 or better).

#### **c) Rotation test.**

**d) Preferential looking** (child fixes more at pattern than homogenous stimulus).

#### **e) Opto-kinetic nystagmous.**

#### **f) Visual evoked potential.**

**2) Children at age of 2 years:** Picture naming test (kay single picture test).

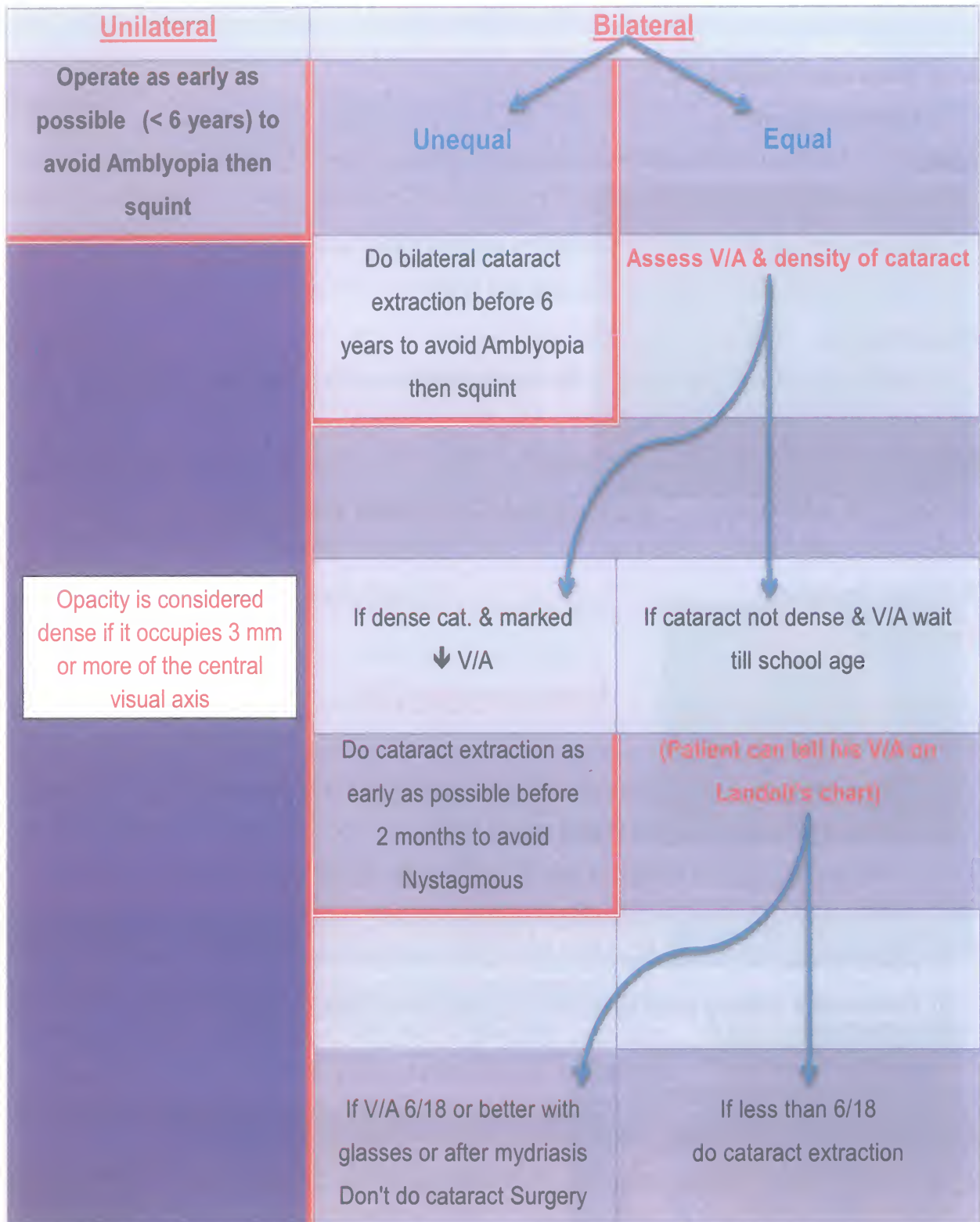
**3) Children at age of 3 years:** Matching single letter opto-type (Sheridan card).

**4) Children at school age:** Landolt's chart.





## Plan of treatment



However, early operation (before the age of 6 m.) is risky due to

1) A.C is shallow

2) Pupil will not dilate properly (dilator ms. is not well developed.)

## **Lines of treatment:**

- 1) Surgical ttt.
- 2) Correction of aphakia.
- 3) Correction of amblyopia.

### **1) Surgical treatment:**

#### **a) Irrigation - Aspiration:**

- ❖ Through 2mm limbal incision, an anterior capsulotomy is done.
- ❖ This followed by Aspiration of lens matter with simultaneous irrigation with saline using double way canula.
- ❖ The central part of the posterior capsule must be removed followed by anterior vitrectomy because the infantile posterior capsule mostly opacity.

#### **b) Lensectomy:**

- ❖ In which the lens is removed (using the vitrectomy machine):
- ❖ Advantage: No posterior capsule opacification,
- ❖ Disadvantages: RD (more common).



### **2) Correction of aphakia:**

- ❖ **Aim:** To avoid amblyopia.

- ❖ **Methods:**

a) Glasses.

b) Contact lenses.

c) IOL.

- ❖ **But:**

a) Glasses

- 2 pairs.
- Heavy.
- Constricted field.

b) Contact lens:

- Difficult insertion & removal.
- Complication (corneal ulcer).

c) I.O.L:

- Power changes with age.
- This explains why 6/18 with accommodation is better than 6/6 without it.

# Senile cataract

## Definition:

- ❖ It is bilateral, gradually progressive lens opacity.
- ❖ Affecting old people.
- ❖ Not suffering from local or general disease.

## Incidence:

- ❖ **Age:** Above 50 years.
- ❖ **Sex:** Equal (m = f)
- ❖ **Hereditary:** May play a role
- ❖ **Side:** Bilateral (but one eye precedes the other )

## Etiology:

- ❖ Unknown, but the metabolic disturbances may be due to:
  - 1) Disturbed capsule permeability.
  - 2) Disturbed pH.
  - 3) Disappearance of metabolically important substances: Vit.C & Glutathione.
  - 4) Endocrinal disturbances.
  - 5) U.V. rays.

### Whatever the cause, the lens undergoes "2 processes":

- 1) **Coagulation of lens proteins:** as decrease pH (due to accumulation of lactic acid) → activated dormant enzymes. (Proteases) → breakdown of lens proteins → amino acid & polypeptides (opacity).
- 2) **Change in the water content** (Hydration or sclerosis).

## Types:

- 1) Cortical 75%.
- 2) Nuclear 20%.
- 3) Cortico-Nuclear.

## Method of examination:

### 1) Oblique illumination:

#### a) By:

- ❖ Torch. Or
- ❖ Slit lamp: with powerful illumination & Magnification, So → gives more details.

#### b) Aim: to examine:

- ❖ The lens opacity,
- ❖ The lens capsule.
- ❖ The anterior chamber depth.
- ❖ The iris shadow.



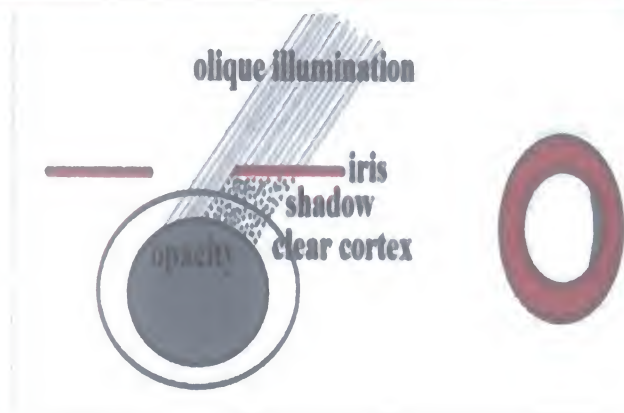
## Iris shadow:

### Definition:

- ❖ It is the shadow of the iris on the lens opacity.
- ❖ **Shape:** Black (dark) crescent in the pupil.
- ❖ **When?** It is present in immature cataract & becomes smaller as the cataract approaches maturity (approaches the anterior capsule).
- ❖ **Occasionally:** the shadow is seen in hyper-mature stage.

The shadow can only form: if a clear interval is present between iris & the opacity.

- ❖ In immature stage, this interval is occupied by clear fibers,
- ❖ But in hyper-mature stage, it is occupied by aqueous.



### 2) Red Reflex: In the dark room:

- ❖ Clear areas → Red.
- ❖ Opacity → Dark.

### 3) Tonometry: As IOP (may be increased).

## Senile Cortical Cataract

### Stages:

#### 1) Immature stage (the lens is not totally opaque). Showing:

- 1) Sectorial opacities at the periphery of the lens that progress gradually (Incipient stage).
- 2) May be cuneiform (ant. cortical wedges of opacity) Or cupuliform (a sheet in the posterior cortex).
- 3) The last fibers to be affected are the anterior sub-capsular lens fibers.



### Symptoms:

- 1) Gradual drop of vision (from  $\frac{6}{6}$  - C.F).
- 2) Fixed musca.
- 3) Uni-ocular diplopia (Due to sectorial changes in R.I.).
- 4) Night Blindness, as the peripheral opacity interferes with the entrance of rays to the peripheral retina.
- 5) Glare & Colored halos around light.

### Signs:

#### 1) Oblique illumination:

- a) Lens → Shows Grayish white sectors.
- b) A.C. depth → Normal
- c) Capsule → Normal.
- d) Iris shadow → present

#### 2) R.R.: Black sectors against a reddish background

#### 3) Tension: Normal.

### **2) Mature stage** (The lens is totally opaque).

Symptoms: Decrease of vision (Hand movement H.M.)

### Signs:

#### 1) Oblique illumination:

- a) Lens → Is uniformly grayish white.
- b) A.C. → Normal depth
- c) Iris Shadow → Absent.

#### 2) Red reflex: Absent.

#### 3) Tension: Normal.

### **3) Hyper-mature stage**

(The lens loses water & shrinks).

Symptoms: Drop of vision (H.M).

### Signs:

#### 1) Oblique illumination:

- a) Lens → Totally opaque
- b) Capsule → Thickened, wrinkled with white ( $\text{Ca}^{++}$ ) & yellow (cholesterol) deposits
- c) A.C. → Deep
- e) Iris Shadow → May be present,

#### 2) Red Reflex: Absent.

#### d) Iris → Tremulous.

#### 3) Tension: May be increased.



## Complications:

1) Leakage of degenerated (irritant) lens proteins: leading to:

- a) Phacotoxic uveitis.
- b) Phacolytic glaucoma: The leaking ptn will be engulfed by macrophages.

The swollen macrophage may get trapped in the TM leading to 2<sup>nd</sup> glaucoma.

2) Degeneration of zonules: Leading to lens displacement (Subluxation or dislocation).

## Types:

1) Typical Hyper-mature cataract: As before.

2) Morgagnian type:



In which, the lens doesn't shrink & the degenerated lens matter becomes liquefied into a milky fluid in which the nucleus sinks by gravity to the lower portion of the lens.

## Intumescent Cataract

❖ In which the process of hydration is suddenly exaggerated.

(Due to rapid breakdown of lens ptn → rapid in osmolarity).

### Symptoms:

- 1) Rapid decrease of vision.
- 2) Pupillary block (in predisposed eye) → ↑ IOP → Ocular pain & Headache.

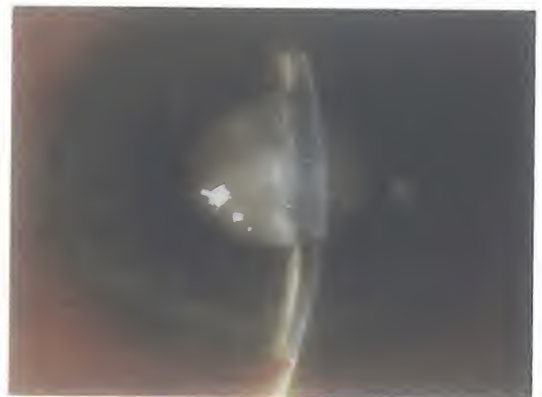
### Signs:

1) Oblique illumination:

- a) Capsule → Glistening & stretched.
- b) Lens → Shows water vacuoles.
- c) A.C. → Shallow.
- d) Iris Shadow → May be present.

2) Red reflex: may be seen (small areas appear red).

3) Tension: may be ↑ (in predisposed eyes) (phacomorphic glaucoma).





	Immature	Intumescent	Mature	Hyper-mature
Symptoms of Vision	↓	↓↓ (C.F., H.M.)	↓↓↓ (H.M.)	↓↓↓ (H.M.)
Signs:				
<b>1. Ob. Illumination</b>				
❖ Lens opacities	Sectorial	Sector + H <sub>2</sub> O	Total	Shrunken
❖ Capsule	Normal	Glistening	Normal	Thickened
❖ AC	Normal	Shallow	Normal	Deep
❖ Iris.	Normal	Pushed forward	Normal	Tremulous
❖ Iris Shadow	Present	May be present	Absent	Absent
<b>2. R.R.</b>	Seen	Seen	Absent	Absent
<b>3. Tension</b>	Normal	May ↑	Normal	May ↑

## Senile Nuclear Cataract

**Definition:** It is pathological lens sclerosis in which the lens transparency is affected.

### Characteristics:

1) Very slow (In progression).

2) The lens may acquire a color (Early → yellow & late → Brown)

(Due to melanin deposition, derived from a. a tyrosine).

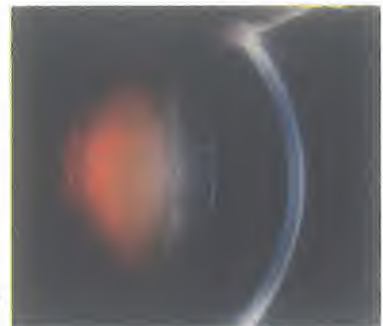
**Grades:** According to the color of the nucleus:

**Grade 1** → Grey.

**Grade 2** → Yellow.

**Grade 3** → Light brown.

**Grade 4** → Brown or black (cataracta Nigra).



### Clinical picture:

#### Symptoms:

1) Decrease of vision due to:

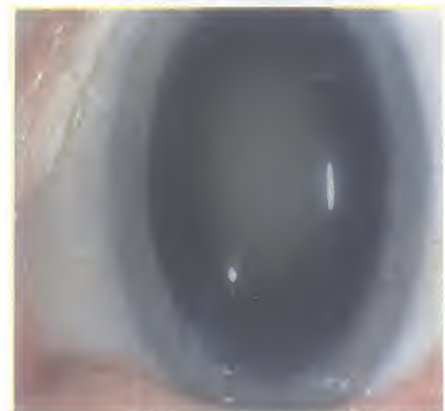
- Opacity.
- Increased R.I. (index myopia).

2) Fixed musca.

3) Uni-ocular diplopia.

4) Day blindness.

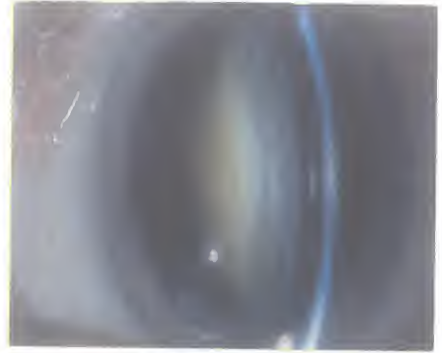
5) **2<sup>nd</sup> Sight:** Process of nuclear cataract causes increase in R.I. of the lens and there for increases the R.P. This makes the pt. myopic. (The pt. can read without his reading glasses).



## Signs:

### 1) Oblique illumination:

- a) Central lens opacity.
- b) Capsule → Normal.
- c) A.C. → Normal depth.
- d) Iris Shadow → Present.



2) R.R: Dark central disc in a reddish background. (Black reflex in cataracta nigra).

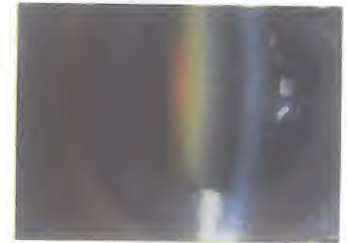
3) Tension: Normal.

## D.D.:

### 1) From senile nuclear sclerosis:

❖ Which is physiological lens sclerosis that doesn't interfere with the lens transparency. By:

- **Red reflex:** normal in S.N sclerosis.
- **Side:** Bilateral & symmetrical in SN sclerosis.



### 2) Senile cataract:

Whether cortical or nuclear from other causes of gradual painless decrease V.A.

(Primary open angle glaucoma – Age-related macular degeneration – Primary optic atrophy)

## Treatment of senile cataract

### Surgical → lens extraction

### ↓ Type of operation (how?)

- 1) Extra-capsular cataract Extraction (ECCE).
- 2) Intra-capsular cataract Extraction (ICCE).
- 3) Phaco emulsification.



### ✚ Time of operation (when?)

- 1) If Phaco technique is decided the earlier the surgery the better.
- 2) If ECCE technique is decided do the surgery when the patient is unable to do his work irrespective to the stage.

### ✚ Which eye to start with?

- 1) The more advanced eye: except if there is intumescent cataract.

1. ECCE (Standard technique)	2. ICCE
❖ Limbal incision.	❖ Limbal incision.
❖ Capsulectomy. (Removal of a circular area of ant. Capsule).	❖ Peripheral iridectomy.
❖ Nucleus removal: either by:	❖ Lens is extracted within the capsule (cryoextraction).
a) delivery through large section (6-8 mm),	❖ Implantation of IOL. (Anterior chamber as there is no capsular support)
b) Phacoemulsification,	
c) Phaco-laser.	
❖ I/A of lens cortex using double way canula.	
❖ Implantation Of IOL (posterior chamber)	
❖ Closure of the wound.	

### N.B. Phacoemulsification:

- ❖ Peripheral small corneal incision (3.2mm) at the upper limb,
- ❖ Anterior capsulotomy.
- ❖ Emulsification of the nucleus using Phaco machine,
- ❖ Irrigation aspiration of lens matter.
- ❖ Implantation of foldable IOL.
- ❖ Closure of the wound by stromal hydration (Sutureless.)

### N.B. Advantages of Phacoemulsification: The smaller the wound →

- 1) The better the wound healing
- 2) The lesser the astigmatism
- 3) The better the rehabilitation

### 2) Implantation of IOL: To compensate for the error of Ref.



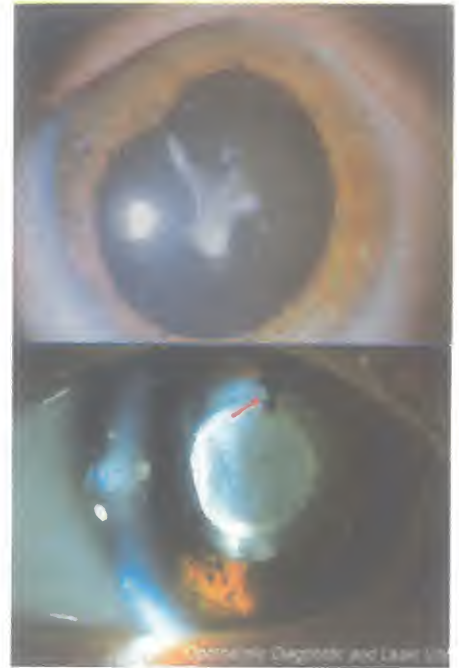
# Complicated cataract

**Definition:** It is lens opacification resulting from local eye disease or general disease.

## **Etiology:**

### 1) Local causes:

- a) Corneal causes: as keratitis
- b) Anterior chamber causes: as glaucoma
- c) Uveal tract causes: as iridocyclitis
- d) Lens causes: as subluxation and dislocation.
- e) Retinal causes: as:
  - ❖ Retinal detachment.
  - ❖ Retinitis pigmentosa,
  - ❖ Chorio-retinitis.
- f) High myopia.
- g) Intra-ocular tumors.
- h) Topical drugs: as steroids, adrenaline and pilocarpine.



## **Mechanism**

The metabolic disturbance is due to: (Diffusion of toxins — interference with lens nutrition)

### 2) Systemic causes:

- a) Diabetes Mellitus: 2 types may develop.
  - ❖ **Pre-senile cataract:** As senile cataract but occurs earlier < 45 yr.
  - ❖ **Snow flack cataract (true diabetic cataract):** In juvenile, uncontrolled DM. The opacities appear first under the anterior and the post capsule as ill-defined flecks, the lens matures rapidly if hyperglycemia is not controlled.
- b) Hypoparathyroidism: May show cataract & tetany.
- c) Galactosemia: Metabolic defect of galactose mechanism produces cataract in the first month of life and is a preventable type of cataract.
- d) Infestation: e.g., anklystoma (common in Egypt) due to:
  - ❖ Anemia (decrease nutrition).
  - ❖ Toxins of the worm.
- e) Systemic drugs: Steroids - Ergot - Amiodarone.
- f) Toxic cataract: Thallium - Naphthalene.
- g) Other diseases: as: Cretinism - Mongolism.
- h) Irradiation: X-ray - Infra-red rays (**glass blowers cataract**) - high voltage electric current.

### Clinical picture:

1) **History:** Of any local or general eye diseases.

2) **Age:** Any age.

3) **General examination:** For any general diseases.

4) **Local examination:**

a) **Lens:**

➤ **Early:** shows:

I. **Posterior cortical opacity:** This site is due to:

- No epithelial lining to the posterior capsule.
- Very thin posterior capsule.

II. **Poly-chromatic luster:** Due to diffraction of the slit lamp light by the granular opacity "cholesterol".

➤ **Late:** shows: chalky white lens (due to Ca deposition).

b) **Signs of a local disease:**

- ❖ Increased IOP (Glaucoma).
- ❖ Posterior synechia (Iritis).

c) **Vision:** is always less than expected from the density of cataract.

d) **Retinal function tests:** often bad.

**Investigations:** e.g. ultra-sonography.

**D.D.:** See table.

	Senile Cataract	Complicated Cataract
1. History	- ve	+ ve (local or general)
2. Age	Above 50 years	Any age.
3. Opacity:		
- Early	Grayish sectors	Posterior cortical.
- Late	Grayish white	Chalky white
4. Eye	Free	May show a local disease
5. Retinal Function	Good	Often bad

### **Managment:**

- ❖ Treatment depends on the cause.
- ❖ Prognosis is usually not good.

#### **1) Local:**

- a) **Iridocyclitis:** Treatment of inflammation first, then 6 month after recovery → Do cataract extraction (under umbrella of steroids).
- b) **Glaucoma:** either:
  - ❖ **2 Separate operations:**  
Glaucoma operation first and 1-3 months later, do cataract op.
  - ❖ **Combined glaucoma cataract operation.**
- c) **High myopia:** Do ECCE (for fear of vitreous loss & R.D.)
- d) **Retinal detachment:** Do ECCE for visualization of the retinal condition.

#### **2) General:** Control the general disease 1<sup>st</sup> then do cataract extraction.

### **The dangers of operation in uncontrolled D.M.**

- 1) Possibility of infection.
- 2) Possibility of Hge (during or after the operation).
- 3) Delayed wound healing.

### **Surgery**

- ❖ Depends upon the **age**.
- ❖ **Soft:** removed by I/A or lensectomy.
- ❖ **Hard:** as senile cataract
- ❖ It is very important to **examine the eye properly** and make sure that the diminution of vision is mainly due to the cataract and nothing else.  
Otherwise, cataract extraction will not be beneficial to the patient.



# Traumatic cataract

**Definition:** It is lens opacification due to Ocular trauma.

## **Etiology:**

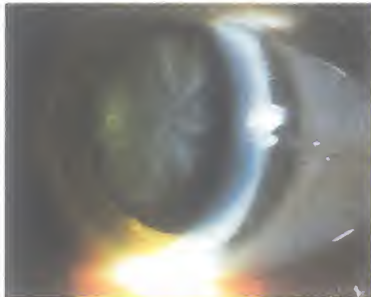
### 1) Perforating trauma (Capsular rupture):

- a) **If small:** (e.g. F.B) leads to localized Opacity.
- b) **If large:** leads to:
  - ❖ Total cataract.
  - ❖ Lens matter may float in anterior chamber
  - ❖ Leading to severe reaction. (**Phaco-anaphylactic-Iridocyclitis**).



### 2) Blunt trauma (Concussion cat.):

- ❖ **Posterior cortical & rosette shaped.**
- ❖ **Mechanism:** Blunt trauma → minute rupture in post capsule allowing aqueous Entry.



## **Vossius ring**

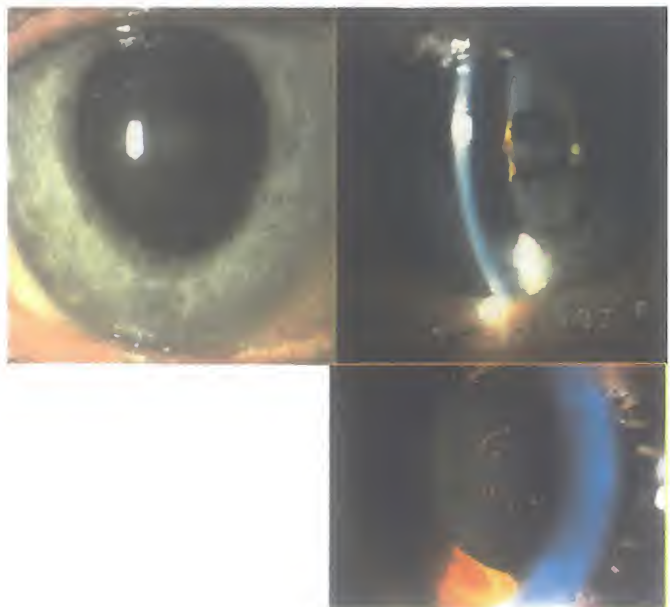
It is a ring of brown pigment on the anterior capsule.  
(Due to impress of the pupillary border of iris on the lens).

### 3) Intra-ocular F.B.:

- a) **Iron F.B.** (sedirotic cataract).
- b) **CU F.B.** (sunflower cat.).

## **Complications:**

- 1) Iridocyclitis.
- 2) 2<sup>ry</sup> glaucoma
- 3) Subluxation & dislocation.



**Treatment:** Meticulous ocular exam. (And US if cataract is dense):

To detect other ocular complication e.g.: vit. Hge. Or RD Or intraocular foreign body.

1) Medical treatment: Topical steroids and atropine.

2) Surgical treatment: Wait till the eye is quite (No I.C or Glaucoma) Then do cataract extraction:

- ❖ If the patient is less than 25 yrs old → I.A. or lensectomy.
- ❖ If the patient is more than 25 yrs old → Phaco or ECCE.

## Unilateral cataract

**Causes:**

- 1) Traumatic cataract.
- 2) Complicated cataract due to local eye disease.
- 3) Congenital cataract.
- 4) Senile (one eye precedes the other).

**Correction of unilateral aphakia by:**

- 1) Contact lens (magnify retinal image size 11%).
- 2) Intraocular lens (magnify retinal image size 1%).

**N.B.: Never by glasses (33%) (Why?)**

The problem is that the aphakic eye will need high plus convex lens which will magnify retinal image unlike the normal eye which has normal retinal image. The difference in the retinal image size (Anisokonia) will cause binocular diplopia.

# After cataract

**Definition:** Opacity in the pupillary area following cataract operation or perforating trauma.

**Composition:** After cataract is composed of:

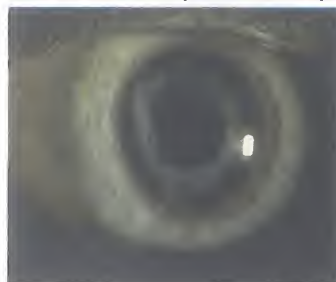
- 1) Part of ant. capsule + post, capsule.
- 2) Remnants of lens matter.
- 3) Proliferated sub-capsular epithelium.

**Clinical Picture:**

- 1) **Opacified posterior capsule:** Late opacification of posterior capsule following cataract extraction (PCO) is not considered after cataract.



- 2) **Sommering ring:** (Opaque ring): Near equator of the lens due to presence of eminent of lens fibers trapped by adhesions between remaining part of anterior capsule and posterior capsule.



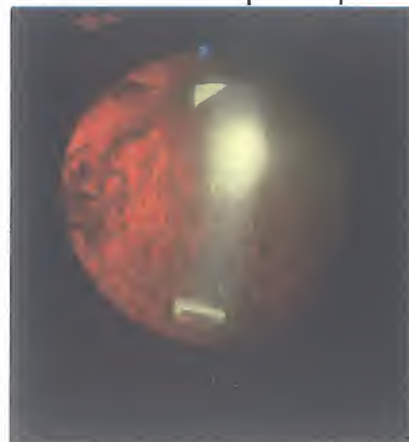
- 3) **Elschnig pearls:** Proliferation of sub-capsular epithelium with formation of transparent pearls (like soap bubbles) in the pupillary area.

**Complications:**

- 1) Iridocyclitis.
- 2) 2<sup>nd</sup> glaucoma.

**Treatment:** If interfere with vision:

- 1) **If thin:** Do capsulotomy either surgical (Using Bowman's needle) or by YAG laser (Cutting laser).
- 2) **If thick:** Do Capsulectomy.





# Aphakia

See later (Errors of refraction)

## Displacement of the lens (ectopia lentis)

**Definition:** Displacement of the lens from its normal position. The lens may be partially displaced (subluxation) or totally displaced (dislocation).

### **Etiology:**

1) **Congenital:** Marfan's syndrome:

- a) Span > Height, Arachnodactyly, High arched palate.
- b) RD, Subluxation.

2) **Acquired:**

a) Trauma.

b) **Degenerative:**

- ❖ Hyper-mature cataract
- ❖ IC.
- ❖ High myopia.
- ❖ Metabolic: Homo-cystinuria.

## Subluxation of the lens

**Definition:** It is partial lens displacement due to partial tearing of the zonules.

### **Clinical Picture:**

#### **Symptoms:**

1) **↓ VA due to:**

- a) Myopia (due to increased lens curvature).
- b) Astigmatism (due to lens tilt).
- c) Complications.
- d) Interference with accommodation.

2) **Uni-ocular diplopia:** If edge of the lens crosses the pupil.



### Signs:

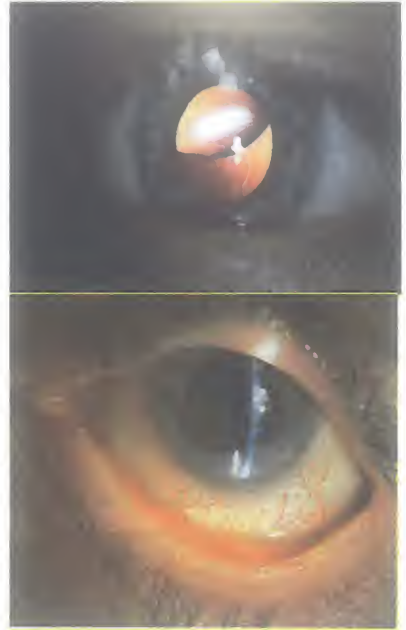
- ❖ AC depth → Irregular.
- ❖ Iris → Tremulous (iridodonesis).
- ❖ Lens → Tremulous (phacodonesis).
- ❖ Edge of the lens may be seen across the pupil.

### Complications:

- ❖ Iridocyclitis → glaucoma.
- ❖ Cataract.
- ❖ Dislocation.

### Treatment:

- ❖ If no complications: glasses to correct the error of refraction.
- ❖ If complications occur: extraction of the subluxated lens either by a scoop plus anterior vitrectomy or by pars-plana lensectomy.



## Dislocation of the lens

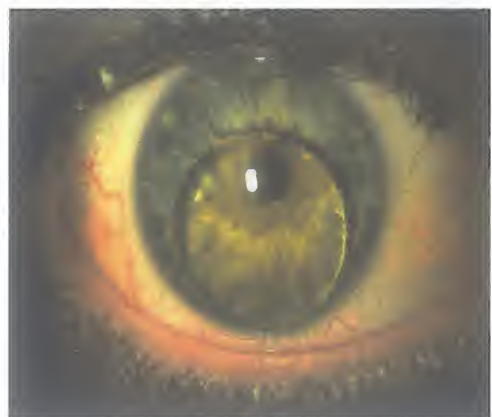
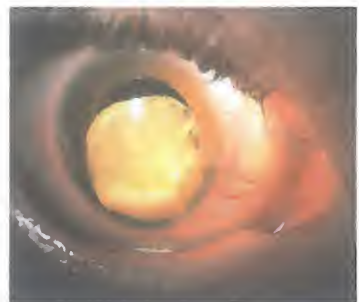
Definition: total displacement of lens due to total tear of zonules

### 1) Anterior dislocation:

- ❖ The lens is present in the AC resembling globule of oil.
- ❖ Fate:
  - Lens → Cataract.
  - Cornea → Endothelial damage which leads to corneal edema.
  - Iris → IC
  - **Glaucoma inversus**: (the patient enters in an attack of glaucoma when the pupil is narrow and the attack is relieved by using mydriatics).

### Treatment:

- 1) Hospitalization.
- 2) Miotic eye drops + anti-glaucoma medication.
- 3) Lens extraction by scooping through limbal incision.



## 2) Posterior dislocation:

### ❖ Clinical picture: Signs of aphakia:

- AC → Deep.
- Iris → Tremulous.
- Pupil → Jet black.
- **Single Perkinji sanson images** (surest sign of aphakia).
- **Lens may be seen** during fundus examination.

### ❖ Fate:

- Eye remains quite if the capsule is intact.
- Uveitis & 2<sup>ry</sup> glaucoma.

### ❖ Treatment:

- If age of pt. ↓ 25 years: Pars plans vitrectomy & lensectomy.
- If age of pt. ↑ 25 years:  
Pars plans vitrectomy + floatation of lens using PFC + Lens extraction by scoop through limbal incision.



# Retinal function tests

- ❖ When visualization of the fundus is obscured by a dense opacity as in mature cataract, or vitreous hemorrhage, the function of the retinal periphery can be roughly estimated using the light projection test.
- ❖ No examination is complete without testing the macular functions in the presence of opaque media.

## 1) Light projection:

- ❖ A strong focused light is used at a distance of 50 cm in a dark room.
- ❖ It is projected to a single eye from the 4 different quadrants to which the patient should rapidly and precisely point.
- ❖ This test gives an idea about the rod function in the retinal periphery

## 2) Macular function tests:

- ❖ Visual acuity, color and form sense are the main macular function tests.
- ❖ In opaque media: the color and form may be tested as follows:

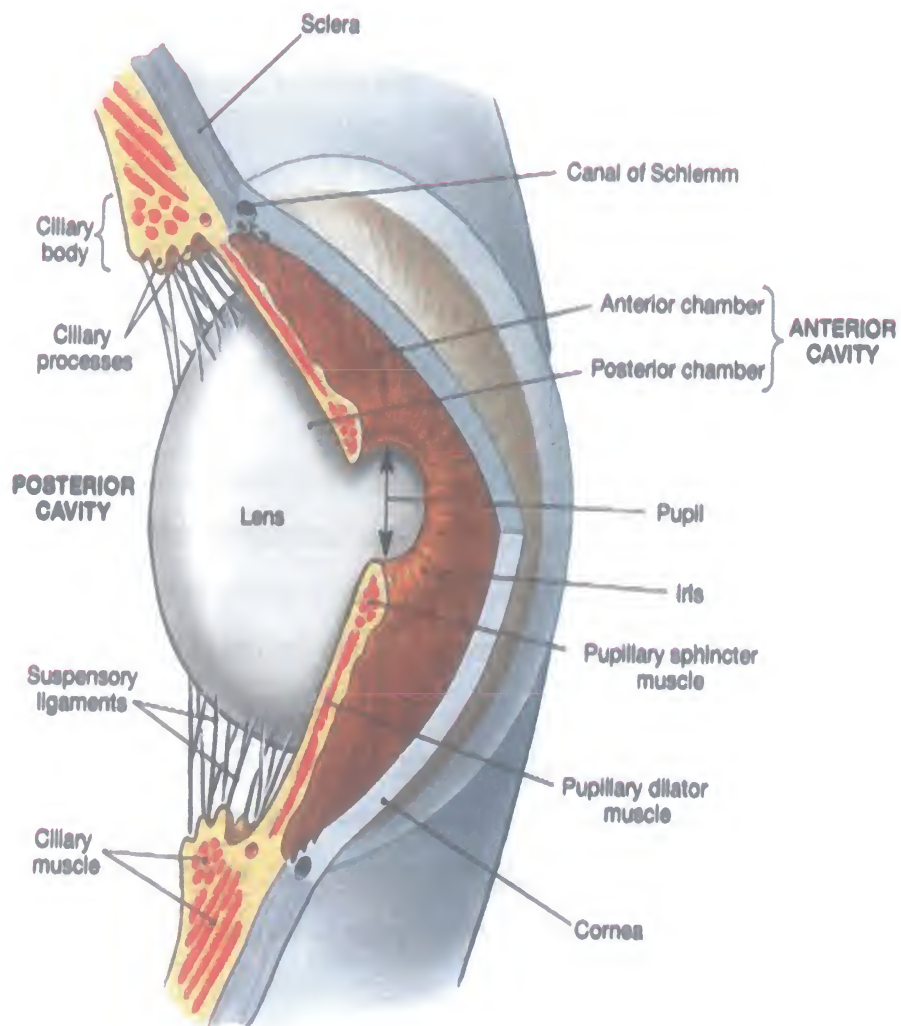
### a) Color test:

- A colored light is projected from a torch or using the color filters of the slit lamp, one eye at time, with the other eye carefully covered.
- If the patient can distinguish between red, and green, then color sense is intact giving a fair idea as the functions of the cones.

### b) Form test:

- An opaque disc is placed in front of the eye to be examined with the other eye covered.
- Holes are made in the center of the disc and a light is put behind the opaque perforated disc.
- If the patient can count the number of the holes, then the form sense is intact giving a fair idea about the function of the cones.

# Uveal Tract



# Uveal tract

❖ It is the intermediate, vascular coat of the eye.

❖ It is divided into:

1) Iris anteriorly.

3) Choroid posteriorly.

2) Ciliary body.

## Anatomy

### The iris

#### **Minute anatomy:**

❖ It is the anterior part of the uveal tract.

❖ It is a **pigmented circular diaphragm** perforated in its center by **the pupil**.

❖ It divides the space between cornea and lens into anterior chamber and posterior chamber.

❖ It has 2 borders:

1) **Free pupillary border.**

2) **Ciliary border** (which is attached to middle of the anterior surface of ciliary body).

❖ It has 2 surfaces:

1) **Anterior surface:** shows irregularities called iris pattern.

2) **Posterior surface:** is smooth & darkly pigmented.

❖ Iris pattern:

📌 **Definition:** Irregularities present on the anterior surface of the iris in the form of elevations and depressions.

1) **Elevations:** Radial streaks: Corresponding to the radial blood vessels running in the stroma of the iris.

➤ **The Collarette:** Irregular circular ring near the pupil.

2) **Depressions:** Are called (crypts) and are present near the ciliary border.

❖ **Color:** The color of the iris at birth is bluish or grayish and it takes the permanent color at age of one year.



### **Minute anatomy:**

1) **Endothelium:** One layer of flat endothelial cells, absent at crypts, and continuous with corneal endothelium.

2) **Stroma:**

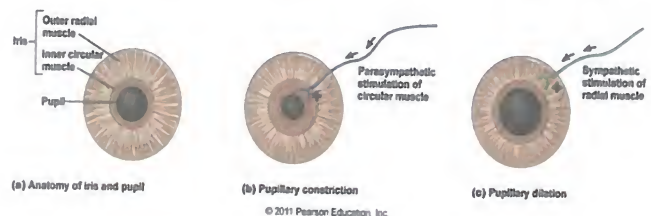
- a) BV (runs radially towards pupillary border).
- b) Nerves.
- c) Pigment cells (melanocytes).
- d) Iris muscles.

3) **Pigment epith:**

- ❖ Two layers of pigmented epithelial cells (ant. flat and post. Cubical) continuous with CB epith.

4) **Iris muscles:**

- a) **Sphincter pupillae:** (constrictor pupillae muscle) Circular band (1mm) around pupil.
- b) **Dilator pupillae:** Radially arranged muscle fibers extending from the pupillary border to ciliary border.



### **Blood supply:**

#### **Circulus iridis major:**

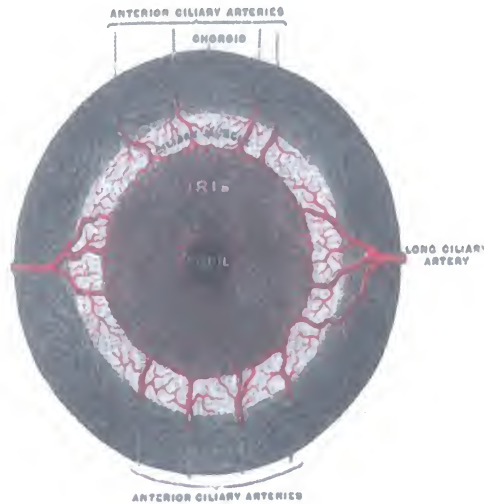
It is a circle of anastomosis present at the base of CB near iris root formed by union of:

- a) **2 long posterior ciliary arteries:** (branches from ophthalmic artery)

Behind the eye and they pierce sclera posteriorly and runs in the supra-choroidal space to reach CB.

- b) **7-10 anterior ciliary arteries:** (branches from muscular arteries which are branches from ophthalmic arteries)

- ❖ This anterior ciliary arteries runs under the bulbar conj. over sclera,
- ❖ One millimeter before limbus, they pierce sclera to reach base of CB where they meet long posterior CA forming circle of anastomosis known as circles iridis major.
- ❖ Branches arising from circulus iridis major runs radially in a zigzag manner from CB border towards pupillary border where they anastomose together forming **circles iridis minor.**



### Nerve supply:

#### ❖ Long and short ciliary nerves passing through ciliary ganglion behind the eye.

- Sensory innervations:** Long ciliary nerves coming from naso-ciliary branch from ophthalmic division of trigeminal nerve.
- Motor innervations:** Mixed short ciliary nerves passing through ciliary ganglion where parasympathetic fibers do relay and sympathetic fibers do not relay.

#### ❖ Parasympathetic fibers → Supply constrictor pupillae.

#### ❖ Sympathetic fibers → Supply dilator muscle.

### Function:

#### ❖ Functions of pupil:

- 1) Control amount of light entering eye.
- 2) Increase depth of focus.
- 3) Prevent light passing through periphery of the lens which acts as a prism (spherical aberrations).

#### ❖ Iris crypts → Aqueous drainage.

## The ciliary body

- ❖ The ciliary body is a triangular body with its base towards the root of the iris and the anterior chamber angle. Its apex merges with the choroid.
- ❖ The ciliary body is composed of two sections, the anterior (**pars plicata**) and the posterior (**pars plana**).
- ❖ The pars plicata carries 70 ciliary processes. Each process has a central vascular core and is covered by 2 layers of epithelium. The ciliary processes secrete aqueous humor, which circulates through the posterior and anterior chambers, playing an important role in maintaining the IOP.

### ❖ The Ciliary Muscle Consists of 3 parts:

- 1) **The longitudinal fibers:** Run between the apex of C.B. and a small lip of the sclera called the scleral spur. Contraction leads to opening of the trabecular meshwork.
- 2) **Circular fibers:** Arranged as a sphincter; contraction causes relaxation of the suspensory ligament of the lens. This relaxes the tension on the lens capsule, causing increased convexity of the lens surfaces and hence increases its power. This action is called accommodation.
- 3) **Oblique fibers:** Are present between the longitudinal and circular fibers.

### **Functions of the Ciliary body:**

- ❖ **Secretion of aqueous humor** from the ciliary processes.
- ❖ Helps **aqueous humor drainage** by contraction of the ciliary muscle.
- ❖ **Suspension of the crystalline lens.**
- ❖ **Accommodation** by the contraction of the circular ciliary muscle.

## The choroid

- ❖ The choroid forms **the major part of the uveal tract.**
- ❖ **It lies between the sclera and the retina**, from the ora serrata to the optic nerve.
- ❖ **This vascular layer supplies nutrition to the external half of the retina** and is composed primarily of an inner layer of capillaries known as the chorio-capillaris, and externally by succeeding larger collecting veins.
- ❖ **Bruch's membrane** is the part of the choroid which lies between the chorio-capillaris and the rods and cones.

**Blood supply:** The choroid is mainly supplied by the short posterior ciliary arteries.

## Diseases of the iris and ciliary body

### **Congenital anomalies:**

- ❖ **Heterochromia:** difference in color between the two eyes.
- ❖ **Anomalies of the pupil:**
  - 1) **Polycoria:** congenital multiple pupils.
  - 2) **Corectopia:** abnormal displacement of the pupil.
- ❖ **Aniridia:** complete absence of the iris, may be with congenial glaucoma,





❖ **Coloboma:** Localized defect in the iris, present since birth and located down and nasally.

The Collarette surrounds the site of coloboma, which differentiates it from an iridectomy.

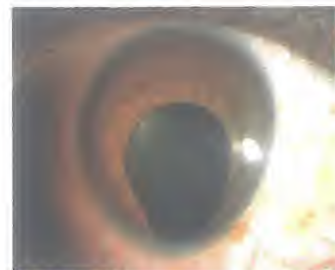
### Inflammation:

❖ **Iridocyclitis** (acute or chronic).

### Tumors:

❖ **Hamartomas** (neurofibromatosis),

❖ **Malignant melanoma.**



## Uveitis

**Definition:** Inflammation of the uveal tract.

### May be:

- ❖ **Anterior uveitis** (iris and anterior part of CB (pars plicata).
- ❖ **Intermediate uveitis** (pars plana and periphery of the choroid),
- ❖ **Posterior uveitis:** choroiditis.
- ❖ **Pan Uveitis:** The entire uveal tract is inflamed.

## Acute Iridocyclitis

### Causes of acute IC:

#### 1) 2<sup>ry</sup> IC:

- a) Keratitis-scleritis.
- b) Lens subluxation – lens dislocation – phacotoxic iritis (hyper-mature cataract) opened anterior capsule following perforating trauma (Phacoanaphylactic iritis).
- c) IOT - IOFB.
- d) Retinal detachment.

#### 2) 1<sup>ry</sup> IC:

a) **Exogenous causes:** Organism enter the eye through surgical wound or traumatic wound causing infective IC.

b) **Endogenous causes:**

#### ❖ **Organism:**

1) Pyogenic in cases of pyaemia, septicemia.

2) Non-pyogenic:

➤ Bacteria: TB – Syphilis – leprosy.

➤ Virus: HSV – HZV.

➤ Protozoa: Toxoplasma.

➤ Round worms: Toxocara.

➤ Fungal organism: Candidiasis.

❖ **Constitutional:**

**1) Diabetes**

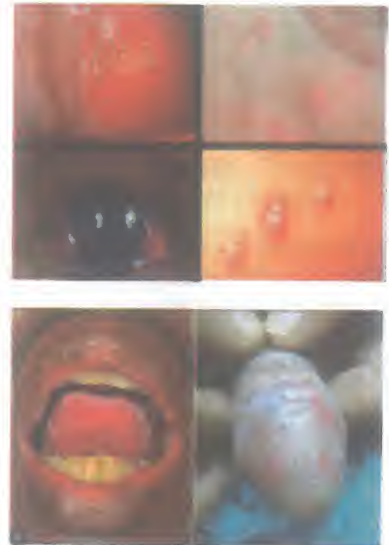
**2) Sarcoidosis:** Multisystem disorder characterized by formation of non inflammatory, non-neoplastic granuloma. 90% of cases → lung infiltration (severity classified according to chest x-ray). Others (skin, eye, CNS).

**3) Arthritis (Spondylo arthropathies) (Ankylosing spondylitis):** Group of overlapping forms of arthritis which are HLAB<sub>27</sub> +ve (i.e. autoimmune disease) characterized by showing also iridocyclitis.

❖ **Other rare disease:**

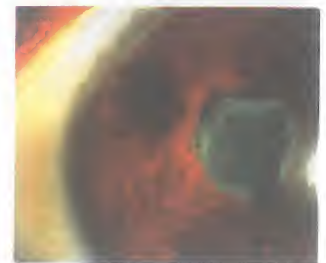
**1) Behcet syndrome:**

- Multisystem disorder.
- Affect young adult.
- Eastern Mediterranean region.
- HLAB<sub>5</sub> +ve
- Lesion:
  - Recurrent oral ulcer.
  - Recurrent genital ulcer.
  - Recurrent IC. + retinal vasculitis.



**2) Vogt Koyanagi Harada syndrome:**

- Multisystem disease
- Southern East Asia (Chinese)
- HLA DR<sub>4</sub> +ve.
- Manifestations:
  - Skin manifestation (alopecia, vitiligo, poliosis)
  - CNS (deafness, convulsions)
  - Exudative choroiditis & iridocyclitis.



**3) Sympathetic Ophthalmitis:**

- ❖ **Bilateral pan uveitis following extensive damage of the uveal tract, following trauma to eye.**
- ❖ **Ag./Ab reaction against the melanin pigment that reaches the blood stream following the trauma.**

## Investigations:

- ❖ 2<sup>ry</sup>: No investigations.
- ❖ 1<sup>ry</sup>: Exogenous: No investigations.
- ❖ Endogenous causes:
  - **Pyogenic organism**: BL culture.
  - **TB**: Chest X-ray, tuberculin.
  - **Syphilis**: VDRL (Venereal disease research lab),
  - **Leprosy**: Wasserman test.
  - **Virus**: Virology studies.
  - **Protozoa**: Compliment fixation test.
  - **DM**: BL Glucose level.
  - **Sarcoidosis**: Chest x-ray & Kviem test (diagnostic),
  - **Bhecet**: (HLA) system studies.

## Clinical picture:

### Symptoms:

#### 1) Pain:

- ❖ Due to irritation of nerve endings of iris by toxins and spasm of ciliary muscles, increase by night due to aqueous stagnation.
- ❖ The pain is: **dull-aching in character (neuralgic)**, referred to the eyebrows and more intense at night.

#### 2) Blepharospasm.

#### 3) Lacrimation.

#### 4) Photophobia.

#### 5) ↓ V/A due to:

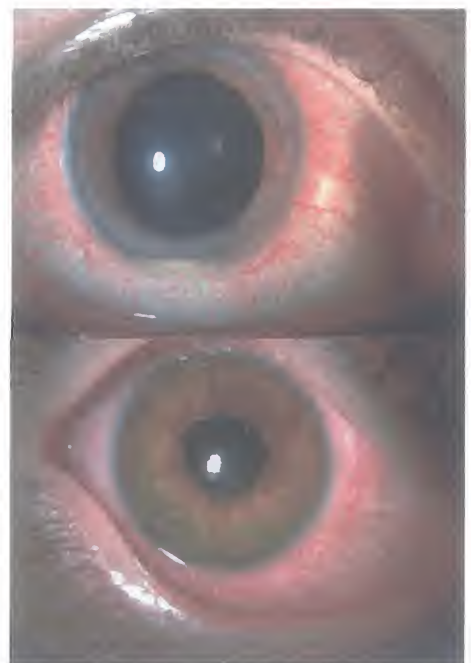
- ❖ **Early:**
  - Cornea Kps: corneal edema
  - AC: Flare- cells-hypopyon & hyphema.
  - Ciliary muscle: Spasm which causes myopia.
  - Macular edema.
- ❖ **Late**: Due to complications.

### Signs:

#### 1) Lid edema.

#### 2) Ciliary flush or injection.

#### 3) Cornea: Kps (keratic precipitates).





## Kps (keratic precipitates)

**Definition:** Inflammatory cells deposited on the back of the cornea due to convection currents as the temperature is 4°C less at the back of the cornea than on the surface of it.

**Site:** On the back of cornea at the lower 1/3 (gravity).

**Shape:**

**Well circumscribed and white:** Recent Kps.

**Irregular and pigmented:** Old Kps.



### 4) AC signs:

a) **Inflammatory cells:** According to the count of the cells detected by slit lamp examination, it can be classified to:

❖ 0-10 → 1+

❖ 21-50 → 3+

❖ 11-20 → 2+

❖ > 50 → 4+

b) **Aqueous flare:** Suspended proteins within the aqueous humor seen using small beam from slit lamp under high magnification, it is classified into:

➤ + → Just detectable.

➤ ++ → Moderately detectable but iris pattern is still clear,

➤ +++ → Moderately detectable but iris pattern hazy,

➤ ++++ → Organized exudates (hypopyon).



c) Hypopyon: Organized exudate at the bottom of AC, it may be:

❖ Sterile.

❖ Suppurative (infected).

d) Hyphema: Causes of hemorrhagic IC:

❖ Traumatic iritis.

❖ DM.

❖ TB.

❖ Viral iritis (HSV, HZV).



5) **Iris:** Muddy iris (loss of iris pattern).

6) **Pupil:** Miosis due to Contraction of constrictor pupillae muscle & dilator pupillae muscle but constrictor is stronger → Straightening of BV due to congestion.

7) **CB:** Tender.

8) **Lens:** Lenticular precipitates & iris pigment on ant. lens capsule.

9) **Vitreous:** Cells & flare.

10) **IOP:** May be high due to plasmoid aqueous or low due to CB shut down.

**N.B.,** Diagnostic signs of acute attack of IC are aqueous cells.

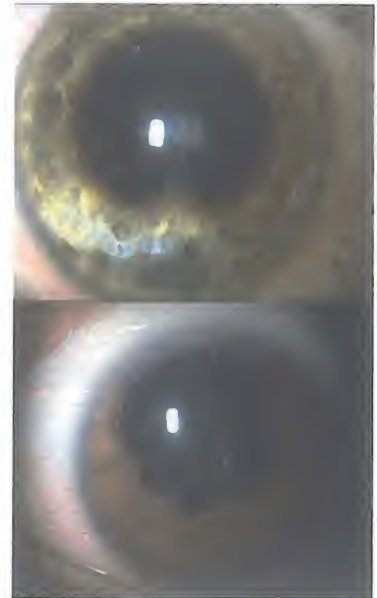
### **Complications:**

#### 1) **Synechia formation: fibrous adhesions.**

a) **PAS (peripheral anterior Synechia)**

b) **Post, synechia**

- ❖ Localized post, synechia Festooned pup.
- ❖ Ring synechia (Succulosis-Pupillae).
- ❖ Occulosio-pupillae.
- ❖ Total post- Synechia.



**How to differentiate between ring synechia and total post synechia**

**The presence of iris bombe → no total post. Synechia**

2) **Complicated cataract** due to diffusion of toxin into lens.

#### 3) **2<sup>ry</sup> Glaucoma:**

- ❖ **Acute:** Due to plasmoid aq.
- ❖ **Late:** PAS & post synechia (ring synechia),

#### 4) **Hypotony:**

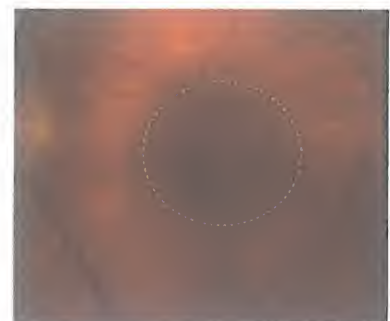
- ❖ **Acute:** CB shut down (shocks).
- ❖ **Late:** CB detachment in cases of cyclitic membrane.

#### 5) **Cyclitic membrane:**

- ❖ Fibrinous membrane in the retro-lental space.
- ❖ Fate: CB detachment, RD (fractional type).

6) **Cystoid macular edema:** Due to the effect of inflammatory mediators on the retinal vessels.

7) **Endophthalmitis, Panophthalmitis.**



### Complications: of longstanding iridocyclitis:

- 1) Band shaped keratopathy.
- 2) Rubeosis iridis and neovascular glaucoma.
- 3) Ectropion uveae.
- 4) Absolute glaucoma.
- 5) Atrophia bulb:

❖ **Definition:** Shrunken organized globe due to cessation of aqueous product.

❖ **Clinical picture:** Small eye – Soft eye (↑ IOP). – Square eye.

(Due to the pressure exerted by the recti muscle on the soft globe).

❖ **Causes:** CB detachment – Atrophic stage of Glaucoma – Long standing RD – Severe perforating trauma.

**Differential diagnosis:** Iritis should be differentiated from other causes of acute red eye.

Symptom	Acute Conjunctivities	Corneal Ulcer	Acute iridocyclities	Acute Congestive glaucoma
Pain	Discomfort	Dull-aching	Dull-aching	Bursting
Vision	Normal	↓	↓	Marked ↓
Discharge	Mucopurulent Or Purulent	Watery (lacrimation)	Watery	Watery
General	Absent	Little	Arthritis & psoriasis	Marked nausea & vomiting
<b>Signs:</b>				
1) lid	Oedema (++++)	Oedema (+)	Oedema	Oedema (+)
2) conj.	Conj injection	Ciliary injection	Ciliary injection	Ciliary congestion
3) cornea	Normal	Ulcer (+ ve fl)	Hazy ( + Kps)	Hazy (Oedema)
4) A.C	Normal	Flare	Flare	Sallow
5) iris	Normal	Normal	Muddy	May be muddy
6) pupil	Normal (RRR)	Normal	Constricted	semi-dilated oval
7) tension	Tn	Tn or T+	T++ or T+	T++
8) fundus	Normal	Normal	Vitreous haze	Not seen

### Treatment:

#### 1) Topical treatment:

##### a) Cycloplgic eye drops:

❖ Atropine sulfate 1%ED.

❖ Dose: 3 times day.

❖ Mechanism:

- Cut already formed synechia.
- Prevent further formation of synechia,
- Relieve spasm of CM ^ pain and headache,
- Decrease surface area of iris → ↓ exudation.



**b) Steroids ED& E. oint:**

❖ **Types:** Prednisolone acetate 1% - Dexamethasone phosphate 0.1%.

❖ **Dose:**

- Every 1 hour until improvement occur then,
- Maintenance dose: 4/day.
- Gradual tapering.

❖ **Mechanism:**

- Anti-inflammatory → Decrease release of mediators via stabilizing lysosomal membrane.
- Anti allergic.
- Anti fibroblastic. (↓ Synechia).
- Anti edema → ↓ Vascular permeability.

❖ **Side effects:**

- ↓ immunity (flare up of infection).
- Comp. cataract.
- 2<sup>ry</sup> Glaucoma (genetically related).

**c) Dark glasses** (for photophobia).

**d) Hot fermentation:** Counter irritation – Vasodilatation.

**2) Systemic treatment:**

**a) Systemic corticosteroids:** e.g. Hostacortin tab (5 mg) dose 1-2 mg/kg on 3 divided doses/day + H<sub>2</sub> antagonist (zantac) to avoid peptic ulcer.

❖ **Contra-indication:**

- Peptic ulcer.
- TB.
- HF.
- Hypertension.
- DM.
- Osteoporosis.
- Infective causes of I.C. (as pyaemia, septicemia).

**b) NSAID:**

❖ **Voltaren, Cataflam** either in combination to steroids or when cortisone contra indicated.

**c) Antibiotic:** if the cause is infection.

**d) Resistant cases to steroids:** give immuno-suppressive therapy as (Cyclosporine) inhibit Ag/Ab Reaction.

**3) Causal treatment.**

#### 4) Treatment of complication:

a) Complicated cataract: Cataract extraction after ttt of I.C & eye remain quite for at least months.

b) 2<sup>ry</sup> Glaucoma:

➤ 2<sup>ry</sup> OAG → medical ttt.

➤ 2<sup>ry</sup> AGG → external fistulizing surgery

c) Cyclitic membrane: Pars plana vitrectomy & membranectomy.

## Iridectomy

### 1) Peripheral iridectomy:

❖ Removal of part near ciliary border not reach long pupillary border it is make upwards → (covered by eye lid away from hypopyon).

❖ Indications: In ACG (all stages).

➤ All ext. fistulizing surgery.

➤ ICCE.

➤ AC IOL & aphakia.



2) Sector iridectomy (key hole): Removal of a part near ciliary border and reaching pupillary border.

3) Wide basal iridectomy (obsolete).

4) Bizarre iridectomy: Any size, any site, any shape (tumor, FB impacted).

5) Visual iridectomy:

❖ In exposed parts & continuous w pupil (down nasal) not covered by lid.

❖ Indications:

➤ Central corneal opacity.

➤ Central lens opacity.

Down and nasal → Allowing near vision

### How to differentiate between visual iridectomy, iris coloboma?

1) Coloboma: commonly bilateral while iridectomy: unilateral.

2) History of surgical interference (iridectomy) while coloboma no history

3) Central corneal or lens opacity in cases of iridectomy.

4) Visual iridectomy: collaret interrupted.

5) Coloboma: continuous around the pupil

# Chronic iridocyclitis

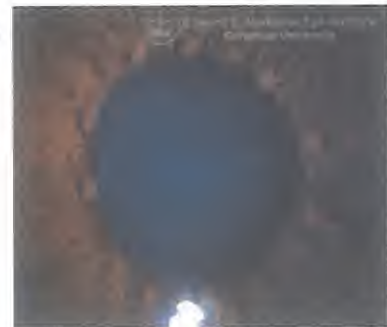
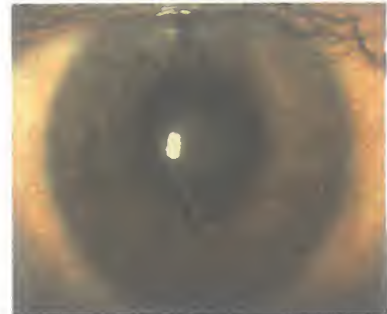
## Etiology:

- 1) Non-granulomatous: Any acute type may turn chronic.
- 2) Granulomatous:
  - a) Tuberculosis.
  - b) Syphilis.
  - c) Sarcoidosis.
  - d) Brucellosis.
  - e) Iritis phacoanaphylactica.
  - f) Sympathetic ophthalmia.

## Characteristic features:

- 1) Long course with exacerbations and remissions.
- 2) Mutton-fat keratic precipitates: (large Kps formed by coalescence of small Kps), beige in color, waxy in appearance.
- 3) Iris nodules:

- a) koepe nodules: at the pupillary margin. →
- b) Busacca nodules: from the anterior surface of the iris.



- 4) Dust-like opacities in the vitreous, which are mainly wandering macrophages.

**D.D.:** Iridocyclitis should be differentiated from other causes of red eye.

(Mainly conjunctivitis corneal ulcers and acute congestive glaucoma).

# Inflammations of the choroid

- ❖ Inflammation of the choroid is called choroiditis or posterior uveitis.
- ❖ Posterior uveitis can affect the retina, retinal vessels, RPE, the choroid, and optic nerve,
- ❖ It may be exudative.

## Granulomatous or Suppurative:

- 1) **Exudative:** a non-specific response with marked exudation.
- 2) **Granulomatous:** due to allergy to bacterial toxins:
  - a) To tuberculo-protein.
  - b) To streptococcal toxins as in a septic focus.



### 3) Suppurative:

- a) Endophthalmitis.
- b) Panophthalmitis.

## Choroiditis

N.B.: There is always retinitis 2° to choroiditis so actually it's Chorio-retinitis.

### 1) Exudative choroiditis

**Definition:** it is non-specific choroiditis with much exudation.

**Pathology:** allergy to endogenous toxins tuberculo-protein – septic focus (streptococcal Toxin).

**Clinical picture:**

**Symptoms:** Retinal symptoms:

- a) Metamorphopsia, micropsia, macropsia (due distortion of photoreceptors).
- b) Photopsia (due distortion of photoreceptors by inflammation).
- c) Scotoma (+ve & later - ve).
- d) ↓ Of vision (due to vitreous haze & retinal oedema).
- e) No pain.

**Signs:**

- a) Recent focus:
- b) Headled focus:
  - ❖ Yellow.
  - ❖ White
  - ❖ ill defined.
  - ❖ Well defined
  - ❖ Vitreous haze & cloudy retina.
  - ❖ Vitreous → clear.

**2) Granulomatous:** Granulomatous reaction with formation of nodule due to

(T.B. → Tubercle Or \$ → Gamma).

**3) Suppurative:** Due to endogenous or exogenous infection:

- a) Endophthalmitis
- b) Panophthalmitis (see orbit).

N.B.: Posterior uveitis differs from anterior uveitis (iritidocyclitis) in being:

a) Painless

b) No marked visual loss

c) 2° glaucoma is not common

# Endophthalmitis

**Definition:** It is acute suppurative inflammation inside the globe with outer coat (cornea, sclera) & Tenon's capsule is free.

## **Etiology:**

- 1) **Acute post-operative endophthalmitis:** is the most common. It may be infectious, 2<sup>ry</sup> to bacteria or sterile by chemicals as powder from the gloves.
- 2) **Traumatic:** with perforating injuries if antibiotics not started early.
- 3) **Endogenous (metastatic):** As in septicemia & meningitis in immuno-compromised patients.

## **Causative organism:**

- ❖ **Bacterial:** (G + ve) staph, aureus, (G - ve) pseudomonas, Proteus, E. coli.
- ❖ **Fungal:** Candida, Aspergillus.

## **Clinical picture:**

### Symptoms:

- ❖ General (FAHM)
- ❖ Severe pain & headache.
- ❖ Hyperemia.
- ❖ Loss of vision (no PL).

### Signs:

- 1) **Lid:** Oedema.
- 2) **Conjunctiva:** Chemosis, (ciliary & conjunctival injection).
- 3) **Cornea:** Hazy with keratic precipitates.
- 4) **A.Ch:** Hypopyon.
- 5) **Fundus:** Yellowish reflex (Amaurotic cat's eye) d.t. pus in vitreous.



To differentiate between bacterial & fungal endophthalmitis, the following criteria should be considered:

### 1) **Bacterial:**

- a) **Sudden onset** (1-7 days postoperatively), rapid progression.
- b) **Severe:** pain, redness & lid oedema.
- c) **Rapid loss of vision.**
- d) **Hypopyon & glaucoma.**

## 2) Fungal:

- a) **Delayed onset** (8-14 days or more).
- b) **Less: pain, redness & edema.**
- c) **Good light perception** in early cases.
- d) **Transient hypopyon** with localized grayish white condensation in the vitreous (snowballs).

## 3) Sterile:

- a) May look like bacterial or fungal infection.
- b) **Foreign body** (sponge filaments, powder) **may be seen inside the eye.**

## **Complications:**

- a) Panophthalmitis.
- b) Atrophia bulbi.
- c) Pseudo-glioma.

## **Treatment:**

### 1) Hospitalization.

### 2) Vitreous aspiration for smear, culture & sensitivity.

### 3) Ant. chamber paracentesis then:

#### ❖ **Early cases:**

##### a) **Medical treatment:**

- Antibiotics: Vancomycin, ceftazidime & quinolones
- Massive doses (systemic, drops, ointment, sub-conjunctivally & intravitreal injection) with vancomycin (G +ve) 7 Ceftazidime (G-ve).
- Atropine 1%: Drops & ointment.
- Steroids: After 24 hours of intensive antibiotics (except if fungi are suspected → give Amphotercin-B).

##### b) **Vitrectomy:** in controlled cases with V.A. = PL.

#### ❖ **Late cases:** Evisceration.



# Panophthalmitis

**Definition:** As endophthalmitis but including outer coat, Tenon's capsule & soft orbital tissues.

**Etiology:** As endophthalmitis.

N.B. Endogenous infection usually → endophthalmitis as organism is attenuated by circulating

Abs BUT exogenous infection usually → Panophthalmitis due to large number of virulent organisms.

**Clinical picture:** As endophthalmitis + Signs of affection of Tenon's capsule:

- a) Proptosis.
- b) Limitation of ocular movement.

**Complications:**

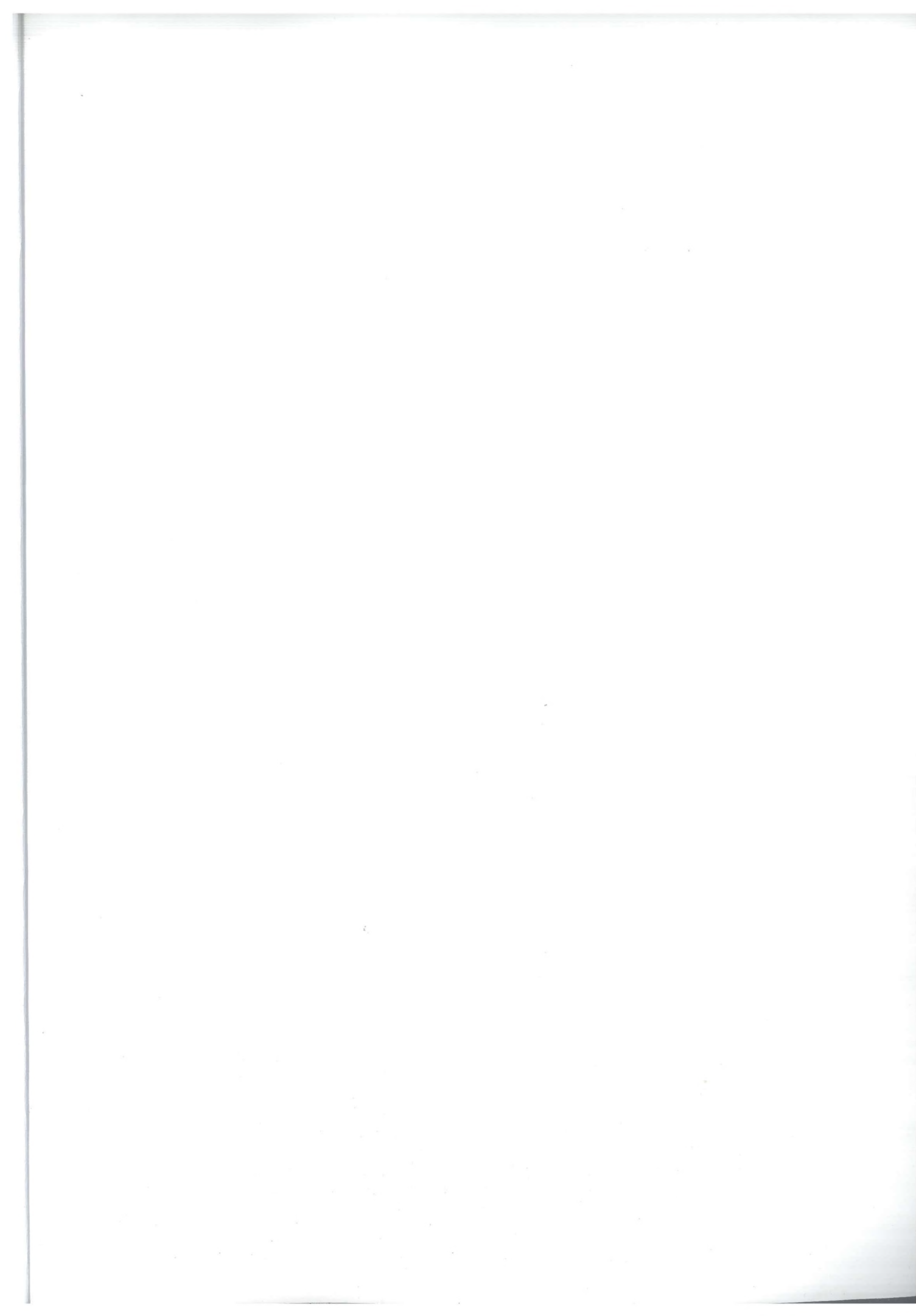
1) Atrophia bulbi (pus may burst through cornea).

2) Spread of infection:

- a) Orbital cellulites.
- b) Cavernous sinus thrombosis.
- c) Meningitis.

D.D.: Endophthalmitis, cavernous sinus thrombosis & orbital cellulitis

**Treatment:** Evisceration.



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2

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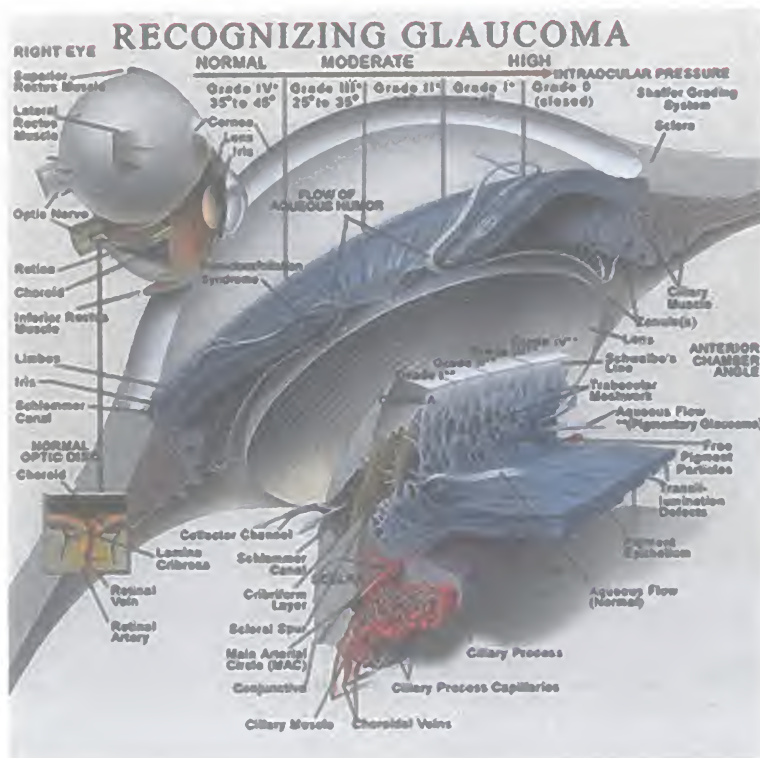


## Index

Glaucoma .....	1
Retina .....	29
The Vitreous .....	55
Optic nerve .....	57
Neuro-ophthalmology .....	69
The orbit .....	75
Eye injuries .....	89
Errors of refraction .....	107
Pediatric ophthalmology & strabismus .....	121
The eye & systemic diseases .....	137
Ocular tumors .....	144
Ocular pharmacology .....	149
Lasers in ophthalmology .....	156
Differential diagnosis .....	157



# Glaucoma



# Glaucoma

## Introduction

### Importance of IOP:

- 1) To keep the shape of the retina during eye movements.
- 2) To keep the aqueous humor circulating.
- 3) To supply nutrition to the avascular structures of the eye. (Cornea & Lens).

### 1) Aqueous humor:

**Definition:** Clear fluid present in the anterior & posterior chamber differs from plasma only in its low protein contents. ( $1/200$ ) of that plasma.

N.B. In cases of inflammation → protein contents in the aqueous humor increases and so called **plasmoid aqueous**.

### Production of aqueous humor:

By outer non pigmented C.B. epithelium lining ciliary processes.

N.B. Each process is made of stroma wide capillary covered by 2 layers of epithelium.

(Outer non pigment and inner pigmented)

### Mechanism of aqueous production: (Two step mechanism)

Aqueous production is divided into:

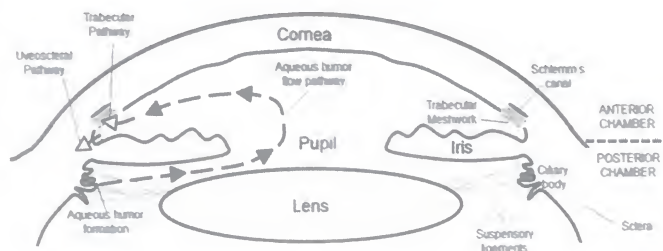
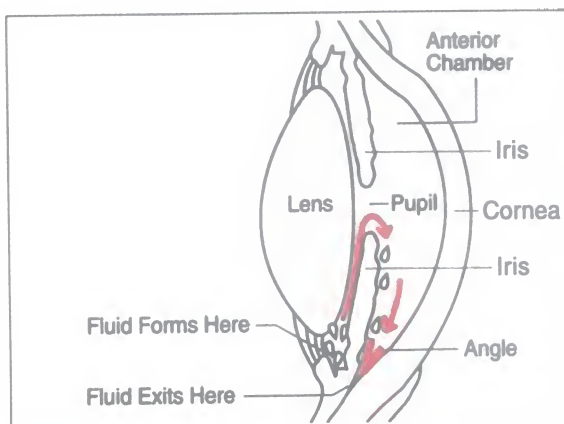
- 1) **Active secretion (80%):** Needs energy & several enzymes such as carbonic anhydrase enzyme.
- 2) **Passive secretion (20%):** Depending on:
  - a) **Ultra filtration:** which is the pressure gradient between the blood in the ciliary processes & IOP
  - b) **Diffusion:** Which depends on osmotic gradient.

### Circulation of aqueous humor:

Ciliary process → Posterior Chamber → Pupil → Anterior chamber → Angle of anterior chamber.

### Drainage of Aqueous humor:

- ❖ 90%: Through angle.
- ❖ 10%: Uveo-scleral out flow through iris crypts & C.B. to supra choroidal space then to vortex veins.



## 2) Angle of anterior chamber:

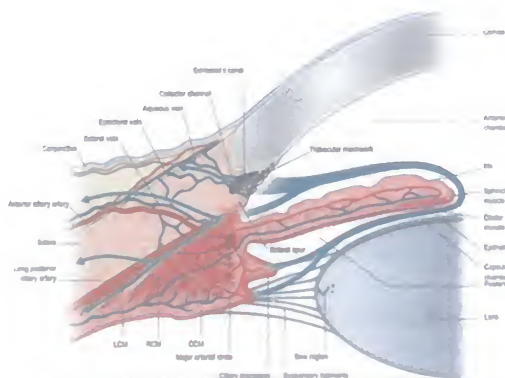
- ❖ **Angle of A.C. can't be examined by direct illumination** as rays coming from the angle of A.C. will always undergo total internal reflection at the cornea air interface.
- ❖ **Total internal reflection occurs** as rays coming from the angle of A.C. forms an angle larger than critical angle.
- ❖ **To overcome this problem, we can examine the angle using contact lens (Gonioscopy lens)** which changes cornea air interface (2 different medias, i.e. 2 different RI) into corner gonio-lens interface (Has the same RI).

### Angle structures are:

1) **Schwalb's line:** It is a rounded border representing end of descemet's membrane (appears white line by gonioscopic examination).

### 2) Trabecular meshwork:

- a) Triangular in cross section.
  - b) Sponge like or thief like structure.
  - c) Apex continuous with Schwalb's line.
  - d) Base continuous with Scleral spur.
  - e) Shows minute opening called spaces of Fontana.
- (Appears broad brown or grey area on gonioscopy)



3) **Scleral spur:** Small part of sclera projecting inside A.C. (Appears white band on....)

4) **C.B. band:** Appears narrow brown band.

5) **Root of iris.**

### 6) Canal of Schlemm:

- a) It is a circumferential canal.
- b) Its outer wall contains openings of collector channels which leads to aqueous veins → Epi-scleral veins → Ophthalmic vein → C.S.



- 1) **Schwalb's line** → (white line).
- 2) **TM** → (brown or grey zone).
- 3) **Scleral spur** → (white band).
- 4) **C.B. band** → (brown band).



- ❖ **Grade 4 (35°- 45°)** → 4 structures are seen (**widely open angle**).
- ❖ **Grade 3 (25°-35°)** → first 3 structures are seen (**moderately wide**).
- ❖ **Grade 2 (20°)** → first 2 structures are seen (**moderately narrow**).
- ❖ **Grade 1 (10°)** → only Schwalb's line is seen (**narrow angle**).
- ❖ **Grade 0 (0°)** → irido-corneal touches.

### 3) IOP:

#### Normal IOP:

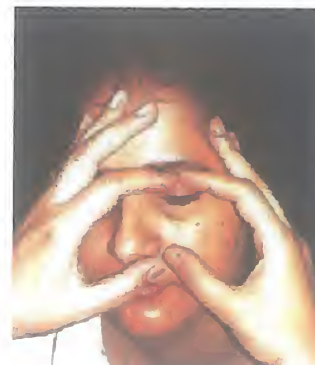
IOP is not a rigid number but it is **continuously changing** just like ABP, being **highest in the early morning and lowest in the evening** and changing with ABP, HR, body temperature and hormonal changes but with certain limits: (Range from 10 mmHg to 22 mmHg).

- 1) **Diurnal variation:** difference between highest and lowest recorded daily pressure **should not exceed 4 mmHg**
- 2) **Difference between the two eyes** **should not exceed 2 – 3 mmHg**
- 3) **Drinking 2 liters of water** then measuring IOP after 30 min **should not record a rise more than 4 mmHg**

#### How can we measure IOP ?

##### 1) Digital method (Technique):

- ❖ Ask the patient to look down but without closing the eye.
- ❖ One index finger gently indents the globe in the upper nasal quadrant.
- ❖ The second index finger placed at right angle to the first pushes into the globe in the upper temporal quadrant.
- ❖ The pressure transmitted to the upper nasal index finger is an estimate of IOP.



## 2) Schoitz indentation tonometer (Technique):

- ❖ With the patient supine.
- ❖ The tonometer is placed perpendicular to the cornea.
- ❖ Plunger indents the cornea by its weight and cornea pushes back with an equal force.
- ❖ This force is read on a scale and converted into mmHg.

N.B.1: The drawback of this technique is the assumption that corneal and scleral rigidity are constant, which is not true, and results are marked under-estimation of IOP in myopes & over-estimation in hypermetropes.

**N.B.2: Measures the amount of indentation by placing certain weight on the cornea.**



## 3) Goldman applanation tonometer (Technique):

- ❖ With the patient seated at the slit-lamp.
- ❖ A cone of diameter 3.06 mm applanates an equal area of the cornea after installing a drop of fluorescein dye and using blue filter light to eliminate the cone.
- ❖ This method is less affected by corneal elasticity and gives accurate estimate of IOP.

N.B. Measures the force needed to flatten certain area of the cornea (3.06).

## 4) Air puff tonometer (Technique):

- ❖ An air-puff from the machine applanates a known area of the cornea.
- ❖ At the exact point of applanation the surface of the cornea becomes a plane mirror that reflects a point light projected onto the cornea to a light sensor.
- ❖ This method is very useful in screening large number of people for high IOP.



## Definitions

- ❖ **Glaucoma:** elevation of IOP which affects health of the eye leading to **optic nerve head** damage & field defect. (Affects perfusion pressure ?)
- ❖ **Ocular hypertension:** Elevation of IOP only without causing damage.
- ❖ **Low tension glaucoma:** Normal IOP + ONH damage & field defect.

## Classification of glaucoma

### 1) Congenital glaucoma.

### 2) Acquired glaucoma:

#### a) Closed angle:

- ❖ **Primary:** where the narrow angle is genetically determined.
- ❖ **Secondary:** where the angle is narrowed by an acquired cause as an intumescent lens or posterior synechia occluding the pupil.

#### b) Open angle:

- ❖ **Primary:** where the cause is still unknown.
- ❖ **Secondary:** where the cause for narrowing of the trabecular pores can be known from examination of the eye e.g.: lens matter, pigment granules, red blood cells.

Ex. 5: Glaucoma may also be classified according to the onset of the disease into:

1) Congenital and developmental glaucoma

2) Acquired glaucoma.

## Pediatric glaucoma

### **Classification:**

#### 1) 2<sup>nd</sup> congenital glaucoma:

- |   |                     |
|---|---------------------|
| a) Neonatal iridocyclitis - aniridia  | b) Aphakic glaucoma |
| c) Birth trauma.  | d) Retinoblastoma   |
| e) <b>Sturge – Weber syndrome:</b> (Raised episcleral venous pressure due to A/V communication)                                     |                     |
| f) <b>Lowe's syndrome:</b> Inborn error of amino acid metabolism leads to mental handicap and eye manifestation - muscle hypotonia. |                     |





## 2) Associated with systemic syndromes:

- a) Congenital rubella.
- b) Lowe's syndrome.
- c) Sturge – Weber syndrome.

## 3) Primary congenital glaucoma:

**Definition:** Elevation of IOP due to congenital anomalies closing the angle.

Affects 1/10000 births and is more common when parents are relatives.

### **Classification:**

- 1) **True congenital (40%):** elevation of IOP during IUL.
- 2) **Infantile glaucoma (55%):** elevation of IOP before age of 3 years.
- 3) **Juvenile glaucoma (5%):** Develop after age of 3 years but before age of 16 years.

### **Etiology (Pathophysiology):**

- ❖ Presence of **abnormal meso-dermal membrane (Barkan's membrane)** occluding angle.
- ❖ **Trabecular dysgenesis** (no spaces of Fontana and iris inserted directly into TM).
- ❖ **Forward insertion of longitudinal CM.**
- ❖ **Failure of splitting of AC angle.**
- ❖ **Absence of canal of Schlemm** which may be a cause or a result.
- ❖ An elevated intraocular pressure before the age of two years causes stretching in the coats of the eye resulting in **globe enlargement**, in addition to **the optic nerve cupping** seen in glaucoma

### **Incidence:**

- ❖ **Sex:** Boys more than girls (2:1)
- ❖ **Side:** More common bil. (75%)
- ❖ **Commonly autosomal recessive.**

### **Clinical picture:**

#### Symptoms:

##### 1) Early:

- a) Photophobia.
- b) Blepharospasm.
- c) Lacrimation (after 2 weeks of life).

##### 2) Late:

- a) Large eye.
- b) Hazy cornea.



## Signs:

1- Corneal Haze (edema): the increase of IOP stroma & epithelial edema.

## 2- Buphthalmos

Definition: Large eye before age of 3 years due to elevation of IOP.

Explanation: As collagen of the outer coat is still immature and can't resist the increased IOP.

→ Expand

Manifestation: Large eye:

### a) Cornea:

- ❖ diameter but ↓ less curvature (flattened)
- ❖ (From birth till 6 months) → less than 10.5 mm
- ❖ At age of one year → each final diameter of (11.75mm)
- ❖ Breaks descemet's membrane associated with aqueous influx healing of this breaks is known as (**Haafin striae**)

b) Sclera: Bluish sclera → Stretched and become thinner showing underlying choroids.

c) A.C: Deep

d) Pupil: Large

e) Iris: Tremulous (lack of proper lens support).

f) Zonules: Stretched & may be torn → subluxation

g) Lens: Flattened & displaced back word.

3- IOP: Elevated IOP measured by Schoitz tonometer

4- Fundus: Glaucomatous optic nerve cupping

5- Refraction: Myopia but less than expected from axial length



**4.0**

- ❖ Each 1 mm increase in axial length → results in -3D myopia
- ❖ But here the amount of myopia is less than this value due to:
  - # Flattening of cornea.
  - # Flattening of lens + its back word displacement.

## Complications:

- ✚ Glaucomatous optic nerve atrophy.
- ✚ Lens subluxation & dislocation.
- ✚ Bilateral cases → Nystagmous & MR.
- ✚ Unilateral Cases → Amblyopia & squint.



**D.D.:**

Cloudy cornea	Large cornea	Lacrimation	Blue sclera	Causes of secondary congenital Glaucoma
Birth trauma.	Megalo-cornea.	Resulting from delayed	Children - infants.	
IU infection (rubella).	High myopia.	canalization of	Congenital myopic.	
Metabolic disease (mucopolysacchoridosis)		Naso - lacrimal duct (congenital dacryocystitis)	Over staphyloma	

**Treatment:**❖ **The treatment is essentially surgical.**

Medical treatment is used only to normalize IOP until surgery is done

❖ **Treatment depends on:**

- 1) IOP measurement.                      2) Corneal diameter.                      3) Angle examination.

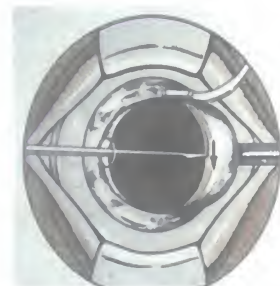
❖ Under general anesthesia using **ketamine anesthesia** not to loose muscle tone.

❖ **Plan of treatment:****1) Corneal diameter less than 14 mm:**

- a) Cornea more or less clear: Goniotomy.  
b) Hazy cornea: Trabeculotomy,

**2) Corneal diameter more than 14 mm:** Ext. fistulizing surgery (subsclearal trabeculectomy)**Surgical treatment:****a) Goniotomy:** It is the surgery of choice whenever feasible

- ❖ Using gonio lens (use goniotomy knife introduced through corneal periphery to cut mesodermal membrane using surgical goniolens)
- ❖ A clear cornea is hence required to be able to perform the operation.
- ❖ Success rate 85%.





### b) Trabeculotomy:

- ❖ In cases where the cornea is hazy, an external approach is used to locate schlemm's canal under a scleral flap.
- ❖ The canal is then cannulated using a curved metal probe (trabeculotome).
- ❖ The probe is then swept in the anterior chamber connecting the chamber to schlemm's canal, hence improving aqueous outflow



N.B. Conjunctival & Trabeculotomy incision is performed following a plane to

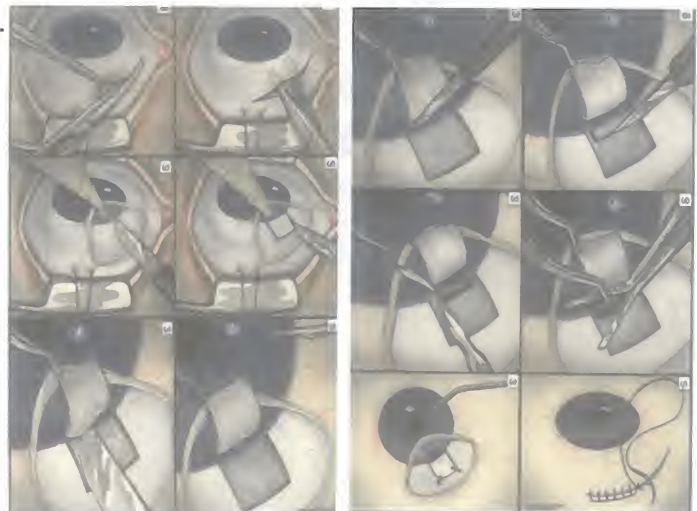
SCHEMATIC CAPSULE

### c) SS Trabeculectomy: (Ext. fistulizing surgery)

- ❖ Is indicated if the above surgeries failed or if the glaucoma is advanced
- ❖ As trabeculectomy but here we **remove block under the scleral flap** which consists of: Canal of Schlemm & TM.
- ❖ Then **peripheral iridectomy** is done.
- ❖ Then **suture the scleral flap** by two 8.0 sutures.
- ❖ The conjunctiva is probably is sutured so that aqueous now passes from:
  - ➡ Posterior chamber → anterior chamber in the TM
  - ➡ Underneath the scleral flap → to sub-conjunctival space → conjunctival Bleb → conjunctival veins.

N.B. SST is done usually with a procedure of Malignant Glaucoma, glaucoma

- ❖ SST is done to **divert aqueous outflow to be drained through conjunctival venous system** as Schlemm canal is obliterated.



### d) Glaucoma drainage: devices (also called tubes or valves) e.g. Ahmed valve

- ❖ These are placed in sub conjunctival spaces and connected to the anterior chamber using a tube which allows aqueous to drain to the sub-conjunctival space.

# Closed angle of glaucoma

✚ **For any reason**, the contact between the lens and iris becomes stronger, the aqueous cannot make its way across the pupil and is trapped in the posterior chamber and pushes against the surrounding structures.

✚ The aqueous will push the peripheral iris forwards (**iris bombe**) until it contacts the peripheral cornea and occludes the angle of the A.C.

✚ Thus, closed - angle glaucoma **start as pupil block that leads to an angle-block**.

## Primary angle closure glaucoma

**Definition:** A condition in which aqueous outflow is obstructed due to partial or completed closure of the angle by peripheral of iris.

### Risk factors:

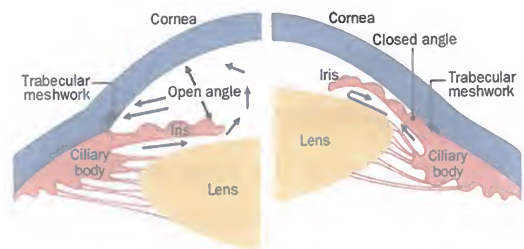
- 1) **Age:** average age of presentation is about 60 years.
- 2) **Gender:** females: males → 4 : 1
- 3) **Race:** more common in southern -eastern Asia.

### Causes:

- 1) **Predisposing factors:** shallow AC & hence narrow angle.

❖ The following factors are responsible for these characteristics:

- ✚ **Lens size:** The lens is the only IO structure that continues to inc. in size throughout life.
- ✚ **Axial length:** Short eyes, which is frequently Hypermetropia  
i.e.: has a shallower AC and also smaller corneal diameter.



N.B. Normal body fluids (especially perimacular fluids) have vasomotor instability leading to a congestion & pushing iris forward!

- 2) **Precipitating factors (Pupillary dilatation):**

❖ Which leads to angle closure (irido-corneal touch) through either:

- a) **Iris bombe theory:** Tight apposition of the iris to the lens (lens become impacted inside the pupil) → pupillary block → aqueous become trapped inside PC → pushing iris mid periphery onward (iris bombe) → angle closure.
- b) **Iris crowding theory:** Pupillary dilatation leads to crowding of the root of iris at the angle.



N.B. The iris bombe (iris bombe) is caused by collapse of the angle closure on doing PI



**Clinical picture of PACG:** PACG is classified into stages:

### **1) Intermittent or sub acute or prodromal stage:**

This stage is characterized by transient attack of papillary block & elevation of IOP which is usually broken after 1-2 hours by physiological miosis → exposure to bright light → sleep

**Clinical picture:** Patient gives history of:

- ❖ Headache & ocular pain.
- ❖ Blurred vision.
- ❖ Colored haloes around light.

**Signs:** In between the attack there are no signs except for:

- ❖ Shallow AC.
- ❖ Narrow angle by gonioscopy (grade 1).

**Fate:** Without treatment patient develop acute attack or pass directly chronic stage of ACG.

**Treatment:** Prophylactic peripheral laser iridotomy.

### **2) Acute congestive stage of PACG:**

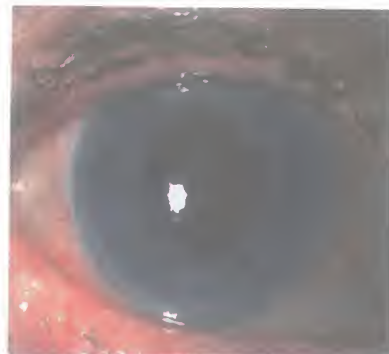
**Clinical picture:**

**Symptoms:**

- ❖ Ocular pain (**bursting pain**) referred to temple.
- ❖ **Blepharospasm - photophobia - lacrimation (reflex).**
- ❖ V/A: **Corneal edema – Optic nerve ischemia**
- ❖ **Nauseas & vomiting** may be occurring due to reflex vagal stimulation.

**Signs:**

- ❖ **Lids:** Edema (due to Blepharospasm).
- ❖ **Conj:** Ciliary hyperemia or flush (due to injection of anterior Ciliary vessels & its branches).
- ❖ **Cornea:** Edema.
- ❖ **AC:** Shallow.
- ❖ **Iris:** Bombe.
- ❖ **Pupil:**
  - **Medilated:** (position at which papillary block occurs)
  - **Fixed:** do not react to light there is nerve ischemia as long as IOP is above 40 mmHg)
  - **Vertically oval** (due to segmental nerve Supply of dilator papillary muscle)





- ❖ **Lens:** show minute opacities known as (Glaucoma flecken)
- ❖ **IOP:** stony hard (digitally) - ↑ 50 mmHg
- ❖ **Fundus:** not seen (due to corneal edema) but if could be seen optic disc will appear at first hyperemia & edematous.

If the patient is in the congestive stage, the IOP will be raised during attack due to corneal edema, and the

lens will be totally closed if the cornea is too thick (grade II).

### Fate:

- ❖ Improve to chronic stage with medical treatment
- ❖ Remain leading to absolute glaucoma.

### Treatment of acute congestive stage

- ❖ Management of acute attack is essentially surgical
- ❖ Medications are given in the pre-operative period for the following goals:
  - a) Lowering the very high IOP to operate under favorable conditions
  - b) Relieving the patient's suffering
  - c) Decreasing intraocular inflammation

**1) Hospitalization:** (Being emergency + if IOP remains 50mm hg for 2 days will lead to OA)

**2) Medical treatment:** for 24 hours to lower IOP before surgical intervention:

a) Systemic treatment: either:

1) Diamox (Acetazolomide): 500mg IV once & then Cidamax 250mg tab 4 times daily.

OR

2) Hyper-osmotic agents:

❖ e.g.:

- **Mannitol 20-25% IV drip:** given 1 - 2 gm/kg
- **Glycerin 50% oral:** (nauseating being sweet): given 1gm/kg
- **Isosorbide 50% oral:** given 1 gm/kg

❖ Mechanism of action: Elevates blood osmolarity and hence: Withdraw water from vitreous (vitreous dehydration) & Decrease passive formation of aqueous.

❖ Administration: Once.

❖ Drawback:

- Elevation of BP.
- Cause vol. over load on the heart.
- Elevates blood glucose level (if glycerin is used, being metabolized into glucose).
- Brain cell dehydration.
- Urine retention in old patient.

b) Topical treatment:

1) Pilocarpine nitrate ED: 1-4% dose every 30 min. until pupil constricts then 4 times daily.

Mechanism:

**Contraction of constrictor pupillae muscle**

1) Causes miosis: therefore relieve pupillary block the iris therefore opens the angle.

2) The stretched iris shows more wide crypts  
→ aqueous drainage

**Contraction of CM**

1) Longitudinal CM:  
Pulls on scleral spur → Widens spaces of Fontana → ↑ Aqueous drainage

2) Circular CM → compresses CB arteries →  
↓ blood flow to CB → ↓ Aqueous formation

N.B. Why we did not start our medication by Pilo instead of systemic treatment?

2) Steroid ED: (Dexamethazone ED) to treat the associated IC.

**If medical treatment succeeds to lower IOP:**

Then → ocular pain is relieved → no corneal edema. Do **Gonioscopy** and if:

1) There is no PAS (peripheral anterior synechia) → then do peripheral YAG laser iridotomy

2) There is PAS → then do ext. fistulizing surgery or SSI

N.B.: If we did ext. fistulizing surgery in such high IOP → expulsive haemorrhage

Therefore medical treatment is given first

**Chronic stage of ACG**

Chronic stage may follow repeated intermittent attacks or acute attack of ACG where the repeated long contact between iris and cornea results in PAS, which results:

a) IOP elevation: IOP is above normal but its value is much more less than during acute attack →  
(no pain → no blepharospasm no lacrimation, nophotophobia, no ↓ VA)

b) ONH cupping.

c) Field defect.

N.B.: Since in a stage asymptomatic shows signs of glaucoma → treat with IOP-lowering

❖ How to differentiate?

❖ Treatment: Ext. fistulizing surgery (sub-scleral trabeculectomy).

## Absolute stage of ACG

**Symptoms:** Blind – painful eye.

**Signs:**

- 1) **Cornea:** Bullous keratopathy (epith bullae) – Corneal ulcer (if bullae rupture).
- 2) **Degenerative pannus Iris:** Atrophic patches ectropion uvea.
- 3) **Lens:** Complicated cataract.
- 4) **Sclera:** Staphyloma.
- 5) **Optic nerve:** OA.
- 6) **Tension:** above 50mm hg.

**Treatment:**

1) **Cyclo-destructive procedure: Destruction of CB by:**

- a) Laser (cyclophoto coagulation)
- b) Heat (cyclodiathermy)
- c) Freezing (cyclocryotherapy)

2) **Retro bulbar injection of 70 % alcohol:**

- ❖ This causes temporary degeneration of ciliary ganglion.
- ❖ Loss of corneal sensation for about 6 months (to be repeated again).

3) **Enucleation** (blind — painful — ugly eye).

Q 8: Peripheral laser iridotomy. It is done at 12 o' clock. Why?

1) To be covered by upper lid to avoid diplopia.

2) If there is hypopyon or hyphema don't obstruct the site of iridectomy.



# Primary open angle glaucoma

## (Glaucoma simplex) (Chronic simple glaucoma)

**Definition:** It's bilateral, genetically determined disease, but not always symmetrical disease characterized by:

- 1) IOP may be high (16% IOP is within normal).
- 2) Glaucomatous optic nerve damage.
- 3) Glaucomatous field defect.
- 4) Open angle seen by gonioscopy.

### **Risk factors:**

- 1) **Age:** more common above 45 years
- 2) **Race:** more common in dark races and difficult to treat.
- 3) **Family history:** strong family history, parents affected: 2x, Sibling: 4x.
- 4) **Myopia:** the two diseases might be genetically linked (myopic patient usually diagnosed unlike other patients)
- 5) **Vaso-spastic disorders:** such as (Migraine, Reynaud's phenomenon) increase risk of normal tension glaucoma.
- 6) **Ocular hypertension:** at high risk to develop OAG.
- 7) **Retinal diseases:** CRVO – retinitis pigmentosa.

### **Clinical picture:**

#### Symptoms:

- 1) Asymptomatic.
- 2) Patients may complain of defective dark adaptation (night blindness)
- 3) Visual field defect which is usually discovered when there is significant field loss.

#### Signs:

(There are cardinal signs in open angle glaucoma)

- 1) Elevated intraocular pressure
- 2) Cupping of the optic disc
- 3) Field changes

## 1) Raised IOP:

- ❖ Measurement of IOP is not diagnostic but important sign
- ❖ IOP within high normal may be deceiving as there is **16% of cases show normal tension glaucoma**.
- ❖ IOP above 21 is suspicious, may not be associated with optic nerve damage and this is known as (**ocular hypertension**).
- ❖ **Asymmetry of the IOP between the two eyes of 5 mmHg or more** should arouse suspicion.
- ❖ Cases of OAG shows **daily fluctuation more than 5 mmHg** in 90% of cases, Therefore it is important to measure IOP at different times during the day (phasing).

## 2) Optic nerve head damage:

### N.B. Anatomical facts to explain CNHD and field defect

Retina is divided by two imaginary lines:-

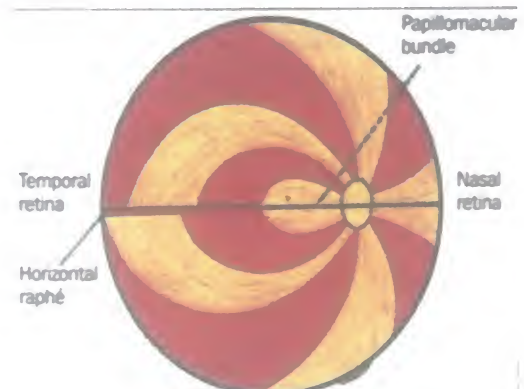
- ❖ Vertical line dividing the disc and retina into nasal and temporal half, fibers from nasal half of the retina must enter through nasal half of the disc.
- ❖ Horizontal raphe or line divides the retina and disc into upper and lower halves.
- ❖ Fibers arising from the macula follow a straight course to the optic nerve head (Forming spindle shaped bundle [papillo-macular bundle]).

### N.B. This bundle occupies most of the temporal half of the optic disc.

- ❖ Fibers arising temporal to the macula follow an arcuate path around the Papillo-macular bundle to reach the optic disc where they are so crowded.
- ❖ Fibers coming from temporal periphery also enter in the upper and lower temporal parts of the optic disc.
- ❖ Fibers coming from nasal retina follow nearly a straight course to be equally distributed in the nasal half of the optic disc.

### N.B. The most crowded fibers are of disc

- 1) Temporal retina other than macula.
- 2) Then of the nasal retina.
- 3) Then of the papillo-macular bundle.



## Theories explaining ON damage

### 1) Mechanical theory:

The elevated IOP posterior bowing of lamina cribrosa openings become no more axial kinking nerve fibers ischemia.

### 2) Vascular or ischemic theory:

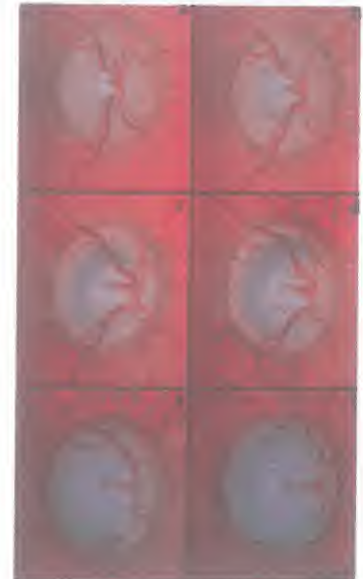
The elevated IOP compromises the micro vessels supplying optic nerve head causing its ischemia.

### 3) Neurotoxicity theory (most recent):

- ❖ Circulating neurotoxins causes alteration of capillary wall tone, which become unable to withstand changes in the IOP (Fluctuation in IOP).
- ❖ High IOP alters the chemical environment of ganglion cell layer forcing them to die (apoptosis) and release cytotoxin to kill nearby cells.

### Early changes:

- a) Large cup disc ratio  $> 0.4$
- b) Asymmetry of C/D ratio between the 2 eyes.
- c) Vertical elongation of the optic cup.
- d) Notching of the rim of the cup.
- e) Splinter hge on the disc.
- f) ↑ Visibility of the bores of the lamina cribrosa.
- g) Nerve fiber layer defects with red free filter.



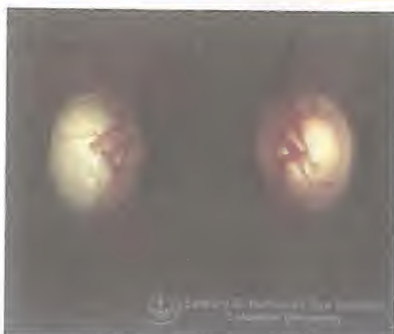
### Late changes:

- a) Large cup with C/D ratio more than 0.7
- b) Deep cup with undermined edges.
- c) Nasal shift of the vessels.

**N.B.1:** In glaucoma progression, the appearance of the optic nerve

should be recorded for future comparison

**N.B.2:** Physiological cupping C/D may be more than 0.3, but it will be bilaterally symmetrical and no progression in the ratio



Normal optic nerve head

Glaucomatous cupping



### 3) Field changes:

#### Changes in the central field (Central 30)

Consists of the papillo-macular bundle and arcuate fibers.

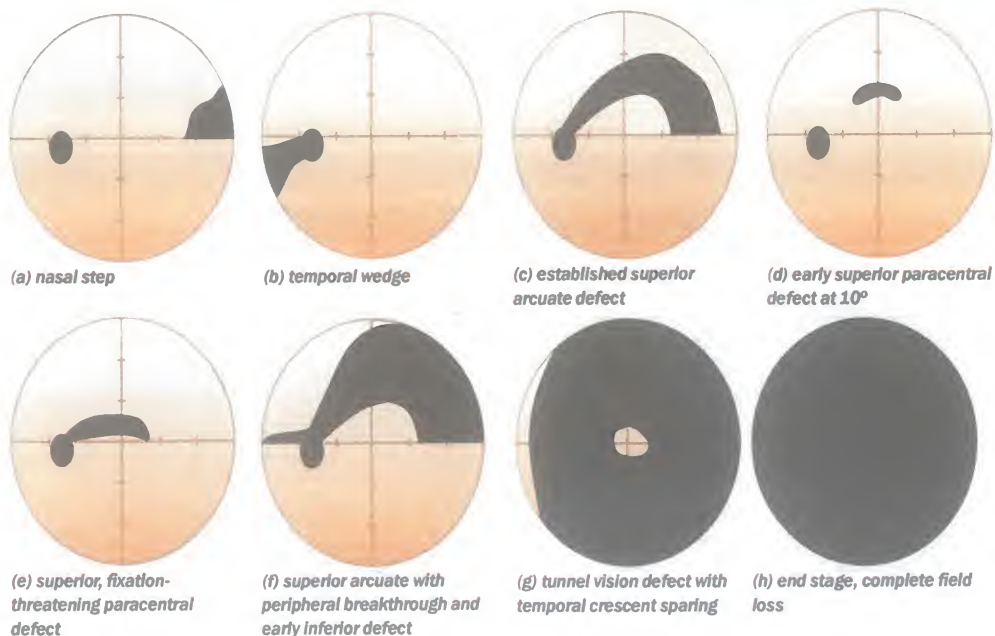
- 1) **Para-central scotoma:** Blind area between  $10 - 20^{\circ}$  of fixation (macular area).
- 2) **Seidle scotoma:** The scotoma elongates to become continuous with blind spot.
- 3) **Arcuate scotoma (BJerrum scotoma):** The scotoma forms an area around point of fixation (affection of the whole arcuate fibers).
- 4) **Ring scotoma (Double arcuate):** When upper and lower arcuate scotoma fuses together.  
( $10 - 20$  degrees around center of fixation)

#### Changes in the peripheral field

- 1) **Nasal step:** Results from asymmetrical shape of the upper and lower field defects as they meet at the horizontal meridian.
- 2) **Nasal contraction.**
- 3) **Temporal wedge**
- 4) **Concentric contraction.**
- 5) **Tubular field:** When the peripheral field defect fuses with the central field.

**N.B.**

- ❖ Till this stage the V/A may be still normal and therefore V/A assessment is not of value in follow up or even in evaluation of open angle glaucoma.
- ❖ Repeated field examination every 6 - 12 months is the ideal method to follow up across of OAG.



#### 4) Optical coherence tomography (OCT):

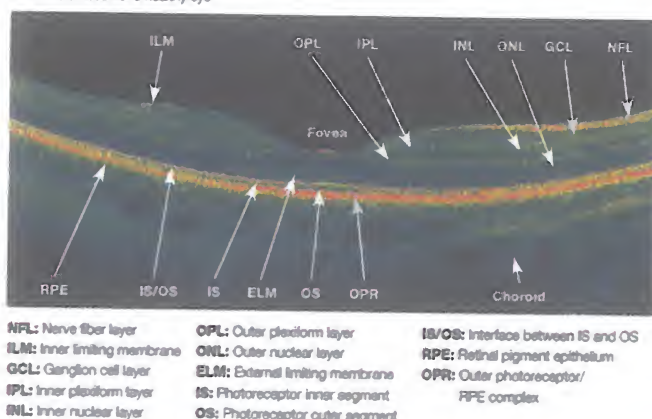
- ❖ Early signs of glaucomatous damage appear in the nerve fiber layer (NFL).
- ❖ Detection of early loss in this layer can help in early diagnosis & follow up of glaucoma before the development of pathological cupping or detectable field changes.
- ❖ OCT uses plane -polarized light to measure the thickness of the nerve fiber layer around the optic nerve head.

### New modalities in diagnosis of OAG

- 1) Heidelberg retinal tomography (HRT): Three dimensional imaging of post. segment of the eye.
- 2) Optical coherence tomography (OCT): Use plane polarized light to measure the thickness of nerve fiber layer around ONH.
- 3) Scanning laser polarimetry: Nerve fiber layer analyzer.
- 4) Double frequency perimetry.



An HD-OCT scan of a healthy eye



### Treatment of OAG

**Aim of treatment:** Is to reach target pressure.

**Target IOP:** Isn't a fixed value but it's the pressure at which no further ONHD or field defect.

It depends on:

- 1) Severity.
- 2) Rapidity.
- 3) Compliance.

**Plan of treatment:**

- 1) Treatment of POAG is essentially medical.
- 2) Start by single medication which is the most effective and more safe and least side effects.
- 3) Treatment either: Medical treatment - Laser treatment - Surgical treatment.

## 1) Medications include:

### a) First choice:

#### 1) $\beta$ - blockers:-

##### ❖ Types:

a) **Non-selective  $\beta$  blockers** ( $\beta_1$  &  $\beta_2$  blockers) [Timolol maleate  $1/4 - 1/2$  %]

b) **Cardio selective  $\beta$  blocker** (Betaxol  $1/2$  %)

❖ Dose: Twice daily.

❖ Effect: Lowers aqueous formation by about 40%.

##### ❖ Side Effects:

1) Worsening of bronchial asthma.

2) Hypotension and bradycardia.

3) Impotence.

4) Masking of sympathetic over-stimulation in diabetic patients when become hypoglycemic.

5) Insomnia.

VB

1) To overcome systemic absorption: Do punctual occlusion for few minutes following installation of the E.D.

2) Few weeks or months: The response to the B-blocker may decrease (Tolerance).

## 2) Prostaglandin Analogues:

##### ❖ Types:

1) **Latanoprost** (Xalatan).

2) **Travoprost** (Traveltan).

❖ Dose: Once daily.

❖ Mechanism: Increase Uveo-scleral outflow (more effective than Timolol).

##### ❖ Side effects:

1) **Hyper pigmentation of:**

a) Lashes.

b) Iris (Heterochromia)

c) Peri-orbital skin.

2) **Eye lashes lengthening.**

3) **Anterior uveitis** (prostaglandins are one of the inflammatory mediators, so not given in patients with history of iritis)

4) **Cystoid macular edema** in aphakics or pseudo-phakics.



## **b) Second choice:**

- ❖ They are added to the above group if IOP is not well controlled.
- ❖ Used if the patient doesn't tolerate the first group.
- ❖ Used for a limited period of a time as post-operative or in secondary glaucoma, because they are usually less effective or their side effects are more frequent

### **1) Alpha agonists:**

❖ **Types:** Brimonidine (ALPHAGAN).

❖ **Dose:** Twice daily.

❖ **Mechanism:**

a) Vasoconstriction of ciliary vessels will lead to decrease in aqueous formation.

b) Increase uveo-scleral outflow.

❖ **Side effects:** Local allergy, dry mouth, fatigue, drowsiness

### **2) Miotics:**

❖ **Types:** Pilocarpine nitrate 1- 4%

❖ **Dose:** Four times daily

❖ **Mechanism:**

1) Contraction of constrictor pupillae will stretch the iris then widens iris crypts and at last will increase uveo-scleral outflow.

2) Contraction of longitudinal ciliary muscle will pull on scleral space and then widens spaces of Fontana.

3) Contraction of circular ciliary muscle will compress ciliary body and will lead to decrease of aqueous formation.

❖ **Side effects:**

#### **1) Miosis:**

a) Exaggerates field defect.

b) If there is central lens or corneal opacity will lead to marked decrease in visual acuity.

c) Permanent miosis even when you stop medication.

d) Decrease night vision.

2) Ocular pain and headache.

3) Iritis.

4) Retinal detachment.

### 3) Topical Carbonic anhydrase inhibitors:

#### ❖ Types:

- 1) **Dorzolarnide** (Trusopt 2%)
- 2) **Brinzolamide** (Azopt 1%)

#### ❖ Dose: Three times daily.

#### ❖ Mechanism: Inhibit carbonic anhydrase will lead to decrease aqueous production.

#### ❖ Side: Allergic conjunctivitis which is more with Trusopt.

### 4) Systemic Carbonic anhydrase inhibitors:

- ❖ These are **very effective medications** but have a lot of **systemic side effects** on the GIT, urinary and nervous system.
- ❖ That is why they are used only for a **short term as in 2<sup>nd</sup> Glaucoma and post-operative cases.**
- ❖ The most common drugs are **Acetazolamide (Diamox – cidamax) 250 mg/6hrs** and **dichlorpheniramide (oratrol) 50 mg/8hrs**

✚ **Start by  $\beta$ -blocker.**

✚ If failed or contraindicated then shift to **prostaglandin analogue** or **carbonic anhydrase inhibitors** or **Alpha Cetamide.**

✚ **If mono-therapy failed give combinations as:**

- 1) **Cosopt** (Timolol + Dorzolamide)
- 2) **Xalacan** (Timolol + Latanoprost) once.
- 3) **Cosopt + pilocarpine.**

## 2) Laser treatment:

- 1) If medical ttt failed to control IOP and therefore filed changes progress.
- 2) Poor compliance.

**Do Argon laser trabeculoplasty**

**Mechanism:** Apply discrete laser burns to trabecular meshwork which causes contraction of the trabecular sheets which will lead to widening of spaces of Fontana.

**Effect:** Lower IOP by 30% only, therefore should not be done if IOP above 35 mmHg.

### 3) Surgical Treatment:

#### Indications:

- 1) If medical and laser treatment failed.
- 2) If medical failed and laser treatment is not available.
- 3) If the patient is unco-operative or the cornea is not clear.

**External fistulizing surgery should be done.**

## Secondary Glaucomas

- ❖ In secondary glaucoma, the rise of IOP is secondary to a local eye disease.
- ❖ In addition to treatment of underlying disease.
- ❖ Beta blocker, local and systemic acetazolamide and hyperosmotic agent may be used until the underlying disease is dealt with to avoid damage to optic nerve and retina.
- ❖ Secondary glaucoma may be of the open or the closed angle type .

## Secondary open angle glaucoma

### Common features:

- ❖ The angle is wide open on gonioscopy.
- ❖ The AC is of normal depth.
- ❖ The cause of glaucoma can be identified by examination.

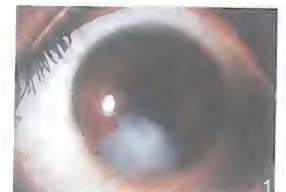
### Classification:

#### **1) Lens-induced glaucoma:**

- a) **Phacolytic glaucoma:** It occurs in **hypermaturation cataract** where the lens protein breakdown into small fragment that leak through the capsule into the AC where they are engulfed by the macrophages. The escaping macrophages are trapped in the trabecular pores. Lens extraction is indicated.



- b) **Phaco-anaphylactic glaucoma:** It follows **trauma or surgery**, where the lens proteins are released into the ocular fluid and initiate an autoimmune reaction. The product of which are deposited on the trabecular pores causing their narrowing and inflammation.





## 2) Glaucoma secondary to intraocular inflammation:

- a) **Glaucomato-cyclitis crisis:** Attacks of unilateral rise of IOP with a normally open angle glaucoma headache and blurring of vision are common due to corneal edema.



The cause is mostly related to disturbance of prostaglandin metabolism in the eye.

- b) **Uveitis:** The cause of glaucoma is plasmoid aqueous as well as swelling of the trabecular pores. The uveitis as usual in addition to beta blockers, local and systemic carbonic anhydrase inhibitors to lower the IOP.
- c) **herpes simplex and zoster:** The causes of glaucoma is inflammation may be complicated with secondary angle closure glaucoma due to occurrence of peripheral anterior synechia.

## 3) Traumatic glaucoma:

Open angle glaucoma following trauma may be due to:

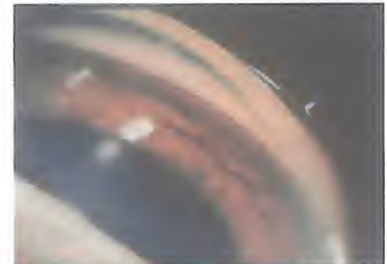
- a) **Traumatic iridocyclitis.**

- b) **Hyphema:** the blood in anterior chamber may obstruct the pores of trabecular meshwork and decrease the aqueous outflow from the eye

- c) **Ghost cell glaucoma:** It occurs in longstanding case of the vitreous hemorrhage where the red blood cell breaks down and their rigid wall (ghost cell) diffuse into aqueous and obstruct the trabecular pores



- d) **Angle recession glaucoma:** glaucoma following trauma secondary to tear of the ciliary body and recession of the angle structures backwards that may obstruct the aqueous outflow channels.



These findings are evident by gonioscopy. →

These findings are evident by gonioscopy. →

## 4) Glaucoma secondary to intraocular tumors:

In choroidal melanoma and retinoblastoma the IOP may rise due to:

- a) The associated intraocular inflammation.
- b) Obstruction of the episcleral veins by the growing tumor
- c) Obstruction of the trabecular pores by the malignant cells

The occurrence of glaucoma depends mainly on the site rather than the size of the tumor

## 5) Corticosteroid induced glaucoma:

Treatment is by stopping steroid therapy, or if steroids are absolutely necessary, topical beta blockers should be prescribed to lower the IOP.

## 6) Miscellaneous causes:

- a) **Pseudo-exfoliative glaucoma:** Secondary open angle glaucoma due to **excessive production of basement membrane-like material by the ocular epithelium**, resulting in deposition of **white dandruff-like material** on the surface of the lens, pupil and trabecular meshwork. Blockage of the trabecular meshwork by this material **obstructs the aqueous outflow from the eye**.



❖ The treatment is the same as primary open angle glaucoma.

- b) **Pigmentary glaucoma:** Secondary open angle-glaucoma due to **liberation of iris pigments into the aqueous humor**, with subsequent deposition in the angle and the back of the cornea (**Krukenberg spindle**).



❖ It affects **middle-aged males with moderate myopia**.

❖ The pigment dispersion results from **excessive rubbing of the peripheral iris against the lens zonules**.

❖ **Transillumination defects of the iris** can be seen in these patients.

## Secondary angle closure glaucoma

In these cases, the IOP rise is secondary to pupillary block or chronic inflammation with subsequent formation of peripheral anterior synechia.

### Common features:

- 1) Gonioscopy shows closure of the angle.
- 2) The AC is shallow.
- 3) The cause of glaucoma can be identified by examination.

### Classification:

#### 1) **Lens-induced glaucoma:**

- a) **Anterior lens dislocation:**

- ❖ It leads to pupillary block.
- ❖ Treatment is by lens extraction.
- ❖ Miotics are contraindicated (glaucoma inversus).

- b) **Intumescent cataract:** The **intumescent lens increases in size markedly** due to its high fluid content. It may encroach upon the anterior chamber, produce a **pupillary block**, or cause angle occlusion, resulting in angle-closure glaucoma.

Treatment is by **cataract extraction after control of the IOP by hyperosmotic agents**.

## 2) Causes in the iris:

- a) **Chronic iridocyclitis:** The cause of glaucoma is **ring synechia or total posterior synechia**. Filtration surgery is indicated.
- b) **Iris tumors or cysts.**
- c) **Pushing the iris forwards** by a rapidly growing posterior segment mass or intra ocular hemorrhage.

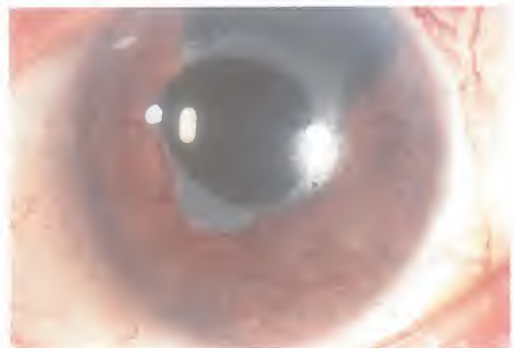
## 3) Traumatic glaucoma:

Secondary angle closure glaucoma following trauma may be due to:

- a) **Traumatic iridocyclitis:** leading to **ring synechia and occlusio pupillae**.
- b) **Anterior lens dislocation:** dislocation of the lens into the anterior chamber.

## 4) Neovascular glaucoma:

- ❖ Neovascular glaucoma is due to **abnormal blood vessels growing from the iris surface to the angle of the anterior chamber**, obstructing the trabecular meshwork and impairing aqueous outflow.
- ❖ **Central retinal vein occlusion** is the most common cause.
- ❖ **It passes into three stages:**
  - a) **Stage of rubeosis iridis:** the abnormal vessels are limited to the surface of the iris. It usually starts at the pupillary border of the iris.
  - b) **Stage of open angle glaucoma:** the abnormal vessels encroach on the angle, with subsequent leakage of proteins that lead to iridocyclitis and subsequent open angle glaucoma.
  - c) **Stage of angle closure glaucoma:** the end stage where anterior synechia closes the angle.
    - In this type of glaucoma miotics are contraindicated to avoid ocular congestion and inflammation.
    - If discovered early, retinal pan laser photocoagulation using argon or diode laser controls the rise of pressure and rubeosis regresses.
    - In neglected cases, cyclodestructive procedures to diminish aqueous formation are indicated. This may be done using trans-scleral diode cyclodestruction.





### Resistant Glaucoma

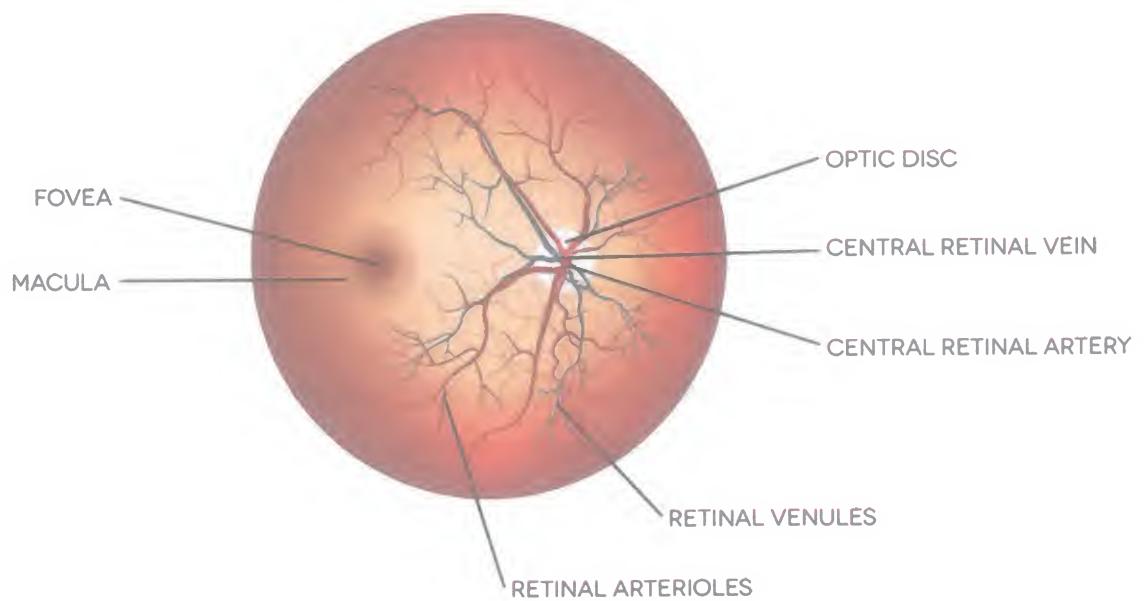
- 1) Congenital Glaucoma.
- 2) Recurrent Glaucoma.
- 3) Pseudo -exfoliation glaucoma.
- 4) Pigmentary Glaucoma.
- 5) Glaucoma in black race.
- 6) Neovascular Glaucoma.

### Treatment: Glaucoma surgery

With Mitomycin or with shunt procedure.

# Retina

## THE RETINA



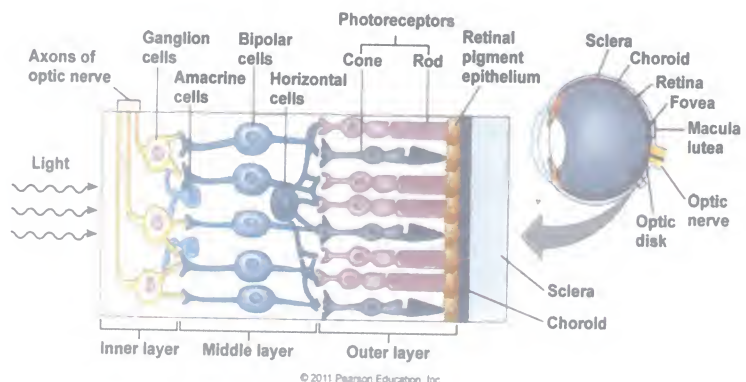
# Introduction

- ❖ Retina is the **inner most layer** of the eye ball.
- ❖ It is the **neuro-sensitive layer** which translates the photon energy to electrical impulses to reach the visual area in the occipital cortex via visual pathway.
- ❖ **Anatomically the retina extends from:**
  - a) **The ora serrata** (Junction between retina and pars plana) peripherally.
  - b) **To the optic disc centrally.**
  - c) **Retina is divided into:**
    - Outer RPE (single layer of hexagonal pigmented cells)
    - Inner sensory retina (9 layers): Varying in thickness from 0.4 mm near the optic disc to 0.15 mm anteriorly at the ora serrata.
- ❖ Separated from RPE by potential space.

**Histology:** The retina consists of 10 layers, which are from outside in

## 1) Retinal pigment epithelium:

- a) Single layer of hexagonal cells containing melanin pigment.
- b) In contact with **Bruch's membrane of the choroid.**
- c) **Adhesions between:**
  - R.P.E and sensory retina are weak (potential space called sub-retinal space)
  - RPE and Brush's membrane are strong.
- d) **Tight junctions** between RPE form the outer blood retinal barrier.
- e) **At the macula:** RPE are taller and contain more melanin and also Xanthophylls-pigment (Macula is darker than the rest of the retina).
- f) **Functions of RPE:**
  - ❖ Nutrition of the outer 5 layers of the retina.
  - ❖ Formation of outer BRB.
  - ❖ Vit. A metabolism needed for visual pigment formation.
  - ❖ Absorption of scattered light.





## 2) Photo receptor layer:

Rods	Cones
Retinal periphery.	Retinal center.
Thin.	Thick.
Night vision.	Day & color.
Rhodopsin.	Iodopsin.

3) External limiting membrane: Formed by the ends of Muller's fibers.

4) Outer nuclear layer: Formed by the nuclei of the rods & cones.

5) Outer plexiform layer: Formed by synapses between (nuclei of the rods & cones) and (the bipolar cells).

6) Inner nuclear layer: Containing the bipolar cells and the cells of Muller's fibers.

7) Inner plexiform layer: Made by the synapses between bipolar cells & ganglion cells.

8) Ganglion cell layer: One layer that increase to several layers at the central part of the retina

9) Nerve fiber layer:

- ❖ Formed by the axons of the ganglion cells.
- ❖ These fibers form the optic nerve.
- ❖ The central retinal vessels are located in this layer.

10) Internal limiting membrane: Formed by the footplates of Muller's fibers & separates the retina from the vitreous.

## **Blood Supply:**

1) Outer layers → By diffusion from choroid.

2) Inner layers → Central retinal artery.

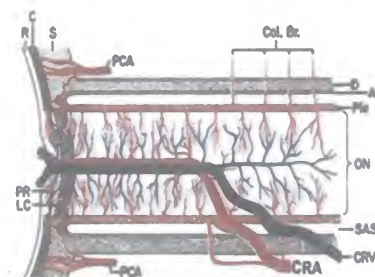
a) CRA is an end artery.

b) Retinal capillaries are present:

- Nerve fiber layer (Superficial plexus)
- Inner nuclear layer (deep plexus)

c) Retina is drained by: CRV which ends in the cavernous sinus.

## Central retinal artery



## Fundus examination

### Methods of examination:

#### 1) By a plane mirror at a distance of one meter:

- ❖ Examine red reflex.
- ❖ Refraction of the patient.

#### 2) Indirect ophthalmoscope using + 20 D lens:

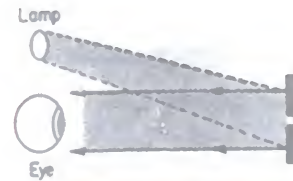
- ❖ **Image of the fundus is:** Inverted.
  - Magnified 3-5 times.
  - Large area can be examined at a time.
  - Binocular.

#### 3) Direct ophthalmoscope:

- ❖ Image of the fundus is:
  - Erect.
  - Magnified 15 times
  - Unocular.
  - Small central area can be examined (Disc + macula).

#### 4) Slit lamp biomicroscopy using + 90 or + 60 D lens:

- Inverted.
- Magnified.
- Binocular.
- Small area seen at a time.



### Structures seen during fundus examination:

#### 1) Optic Disc:

- a) **Site:** Posterior pole (nasal to the macula).
- b) **Diameter:** Vertically oval
  - ❖ **Vertically** 1.8 mm.
  - ❖ **Horizontally** 1.6 mm (average 1.5 mm).
- c) **Color:** Pale pink in the periphery (Neuro-retinal rim) and white in the centre (optic cup).
- d) **Cup:** A pale depression, one third disc diameter (C:D ratio 0.3) from which the central vessels emerge, and in which the dots of the lamina cribrosa may be seen. The normal cup is also called the physiologic cup.
- e) **Edge:** Well defined.



## 2) Retina:

### ❖ Macula:

#### a) Site:

- Temporal to the disc.
- Centre of the macula is 2 disc diameters.
- Temporal and slightly below the center of the disc.

#### b) Diameter: About 7 mm.

#### c) Color: Dark yellowish area. (Why??)

### ❖ Fovea:

#### a) Site: Centre of the macula (it appears as a central depression in the macula as it is thinnest area in the retina being formed of 3 layers only).

#### b) Diameter: 1.5 mm.

#### c) Clinically: Appears as an oval light reflex.

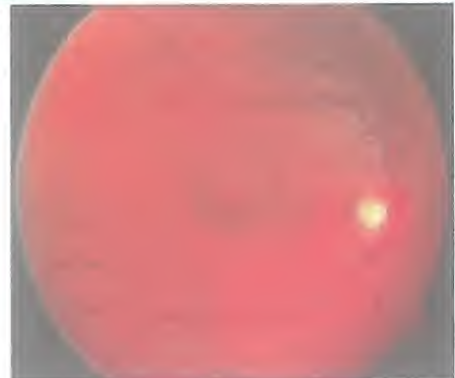
#### d) Contains: Cones only.

#### e) Formed of: three layers only cones and their nuclei (outer nuclear layer).

N.B.: The remaining layers of the fovea are present on its sides (in the surrounding macula).

## 3) FAZ (Foveal avascular Zone):

- a) Lies within the fovea.
- b) 500 µm in diameter
- c) No retinal capillaries.



## 4) Retinal vessels:

- ❖ CRA enters the eye through the cup and divides into upper and lower branches and each divides into nasal and temporal branches.
- ❖ CRV leaves the retina through the cup.

### N.B.: The CRA is:

- ❖ Slightly **nasal to the vein** at the optic cup.
- ❖  $\frac{2}{3}$  of the vein diameter.
- ❖ **Superficial to the vein** at A/V crossings.
- ❖ Sharing **common adventitia with the vein** at A/V crossings.



### 5) Retina proper:

❖ Retina appears pink although it is transparent due to reflection of the color of the blood of the underlying choroid.

a) **Tigroid fundus:** If retinal pigment is not extensive then choroidal vessels with choroidal pigment in between are seen.

b) **Albinotic fundus:** If choroidal pigments are poor (sclera is sum).

## Retinal vascular disorders

**Two basic mechanisms affect the retina in vascular disorders:**

1) **Abnormal vascular permeability**

2) **Retinal ischemia due to generalized arteriolar vasoconstriction**, Prolonged constriction results in hemorrhages, exudates, cotton-wool spots (local micro-infarctions) and lipid exudates

❖ **Vascular Occlusions:**

1) Retinal vein occlusion.

2) Retinal artery occlusion.

❖ **Vascular Retinopathies:**

1) Diabetic retinopathy.

4) Toxemia of pregnancy.

2) Hypertensive retinopathy.

5) Retinopathy associated with blood disorders.

3) Renal retinopathy.

## Central Retinal Artery Occlusions (CRAO)

**Etiology:**

**Embolism:**

✚ **Most common cause of RAO** as: Ophthalmic artery is the first branch from ICA.

✚ **LT eye more common to be affected** as: LT common carotid artery is a direct branch from arch of aorta.

✚ Embolus is usually impacted at the level of lamina cribrosa.

✚ **Sources of embolism are:**

a) **Left side of the heart:**

➤ Calcific emboli valve stenosis.

➤ Vegetations in SBE.

➤ Mural thrombus in cases of A.F.

b) **Aorta** → Fragmented thrombus in cases of aortic aneurysm.

c) **Fat embolism.**

d) **Air embolism.**

## Vaso-obliteration:

### 1) Vessel wall causes:

- a) Atherosclerosis (most common).
- b) peri-arteritis (associated with systemic vascular disorders) as giant cell arteritis and polyarteritis nodosa.
- c) Spasm of CRA → Retinal migraine & Quinine poisoning.

### 2) Causes in blood → Sticky platelet syndrome.

### 3) Causes outside vessel wall → Raised IOP (excessive Ret. buckling).

**Clinical picture:** CRA is an end artery

### Symptoms:

Sudden painless marked decrease in visual acuity except in 15% of cases having a cilioretinal artery as an additional direct branch from ciliary circulation to the macula (tubular field).

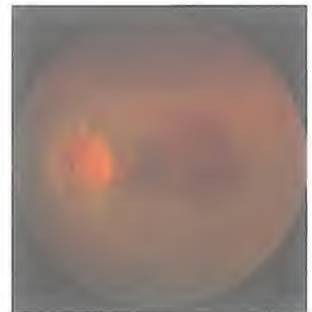
### Signs:

a) **Visual acuity:** Marked decrease in VA (perception of light).

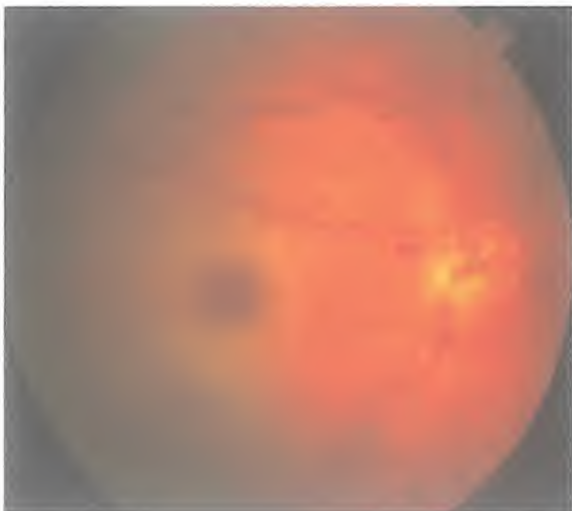
b) **Pupil:** Relative afferent pupillary defect (Marcus qun pupil).

### c) **Fundus:**

- ❖ Arteries: Attenuated.
- ❖ Veins: Segmented.
- ❖ Retina: → Cloudy white in color (due to coagulation necrosis of ganglion cell layer).  
→ Except the Fovea remains red in color (No ganglion cell layer) giving the cherry red spot.



**N.B.: By Fluorescein angiography → Delayed arterial filling**



### **Fate of CRAO:**

#### **↓ Re-canalization of thrombus occurs within days and arterial refilling occurs:**

Cherry red spot disappears (??) → But death of the inner 5 layers and consecutive optic atrophy occurs (V/A → no P.L.) (Pupil → TAPD).

#### **↓ Retinal tolerance time is not yet fixed:**

❖ 5 – 10 minutes.

❖ 90 minutes.

❖ 6 – 8 hours.

❖ More than 8 hours.

### **Treatment of arterial occlusions:**

❖ Start treatment immediately.

❖ Bad prognosis due to short retinal tolerance time.

❖ Patient should lie flat.

#### **1) Lower IOP to dilate retinal arteries, so embolus will dislodge to reach smaller branch.**

➤ Ocular massage for 15 minutes.

➤ Paracentesis.

➤ I.V. CAI (Diamox 500 mg).

#### **2) Vaso dilators:**

➤ Sublingual amylnitrites.

➤ Inhalation of mixture of 5% CO<sub>2</sub> and 95% oxygen.

#### **3) Fibrinolytic therapy:**

➤ Streptokinase.

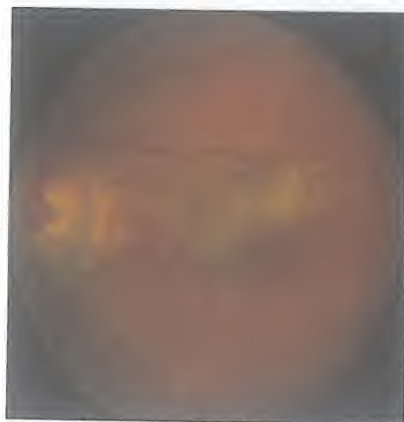
➤ Tissue plasminogen activator: I.V. Or catheterization and injection at ostium of OA.

#### **4) Search for the source of embolism.**

## **Branch retinal artery occlusion**

**Symptoms:** Sudden painless sectorial field loss.

**Signs:** Cloudy white sector of the retina along the course of the occluded artery.





# Retinal Vein Occlusion

## Types:

### 1) Central Retinal vein occlusion (CRVO):

- a) Non-ischemic CRVO.
- b) Ischemic CRVO.
- c) Vasculitis (in young adults).

### 2) Hemi-central vein occlusion (HVO):

- ❖ Due to obstruction of one of the main divisions of the CRV after with the optic disc.
- ❖ May be Ischemic OR Non-ischemic.

### 3) Branch Retinal vein occlusion:

- ❖ Due to obstruction of one of the divisions of the CRV after leaving the optic disc.
- ❖ May be Ischemic OR Non-ischemic.

## Predisposing factors:

### 1) Systemic predisposing Factors:

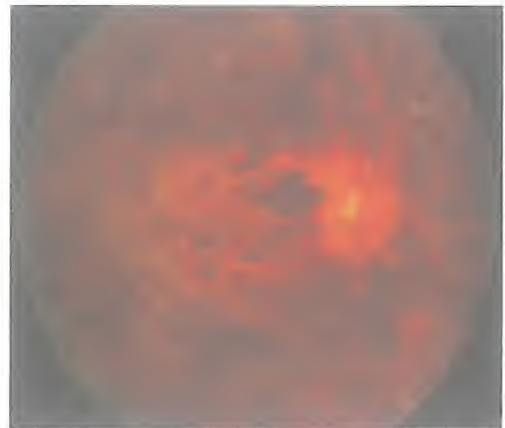
- ❖ Old age (6<sup>th</sup> & 7<sup>th</sup> decade).
- ❖ D.M.
- ❖ Systemic hypertension.
- ❖ Blood diseases (increase viscosity): Polycythemia – Leukemia - Thrombocytosis.

### 2) Local predisposing Factors:

#### a) Wall:

- ❖ Periphlebitis.
- ❖ Cong anomalies of vein wall (Strictures & tortuous course).

#### b) Outside wall → ↑ IOP (1<sup>ry</sup> OAG).



## Pathogenesis

 As a result of vein occlusion:

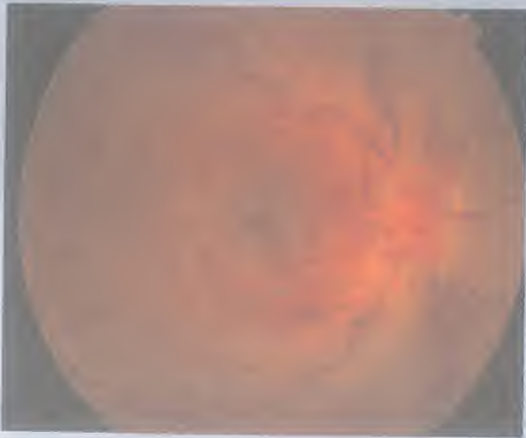

- 1) Veins distal to occlusion: Dilated – congested – tortuous.
- 2) Capillaries: (Leakage): Edema – hge.
- 3) Artery: (stagnation): Ischemia (Cotton wool spots or soft exudates) → Involving nerve fiber layer leading to accumulation of transport material. (Not true exudates)

# CRVO

**CRVO may be:**

- 1) **Ischemic type:** (Site of occlusion before lamina cribrosa).
- 2) **Non ischemic type:** (Site of occlusion behind lamina cribrosa).

	Non-ischemic CRVO	Ischemic CRVO
<b>Symptoms</b>	Rapid moderate drop in V/A noticed usually in the early morning (Why??)	Severe or marked
<b>Signs</b>	❖ Mild RAPD. ❖ Fundus picture.	❖ Marked RAPD. ❖ Fundus picture.

Fundus examination	
Non-ischemic CRVO	Ischemic CRVO
1) Mild dilatation and Congestion of veins	1) Marked
2) Mild retinal edema (macular).	2) Extensive retinal and macular edema.
3) Mild Ret. Hge.(flame shaped and dot shaped Hge).	3) Extensive retinal Hge.
4) Mild disc edema.	4) Extensive disc edema.
5) Few, if present cotton wool spots.	5) Many cotton wool spots.
❖ <b>Prognosis: Good</b>	<b>Poor prognosis due to:</b>
➤ 15% within 4 months.	1) Retinal ischemia.
➤ 30% within 3 years.	2) Neo-vessel formation (NVDS and NVES).
(changes to ischemic type)	3) In severe cases Rubeosis Iridis and neovascular glaucoma.
	

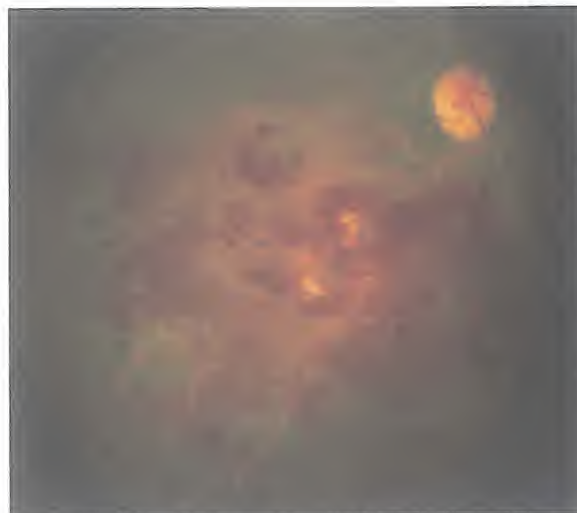
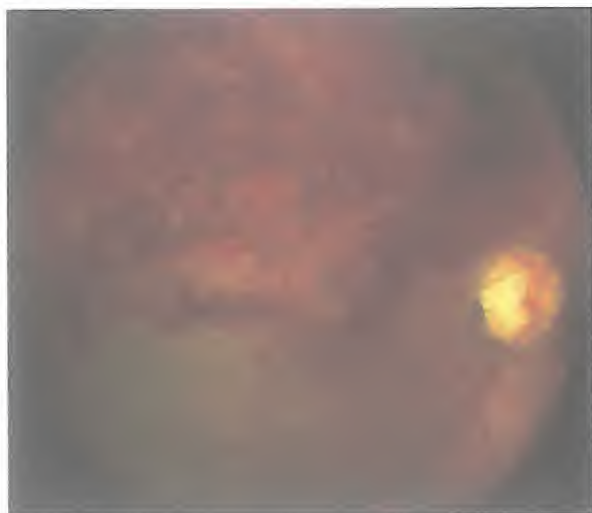
## Management of Central retinal vein occlusion

- 1) Control of hypertension and diabetes.
- 2) After resolution of hemorrhages, **fluorescein angiography** is performed to detect ischemia.
- 3) **Antiplatelet medication** like aspirin 75 mg/day
- 4) **Argon laser photocoagulation** in ischemic cases and in cases with neovascular glaucoma, but only after hemorrhage resolve.
- 5) **Intravitreal drug injection:** Steroids and/or Antivascular endothelial growth factor (AntiVEGFs) for edema and neovascularization
- 6) **Neovascular glaucoma:** is difficult to control and may require:
  - a) Extensive pan retinal photocoagulation.
  - b) Cyclocryotherapy.
  - c) Insertion of a valve as Ahmed valve or Molteno tube.

## Branch retinal vein Occlusion

### Clinical Picture:

- 1) Dilated tortuous congested vein
  - 2) Retinal Hges
  - 3) Cotton wool spots
- } Affecting Sector





# Retinopathies

**Definition:** Bilateral affection of the retina due to a systemic disease

## Diabetic retinopathy

### Introduction:

- ❖ **DM is an endocrinal disease characterized by:** sustained hyperglycemia due to: lack or inefficient endogenous insulin.
- ❖ **There are two main types of diabetes:**
  - a) IDDM – type I – Juvenile M.
  - b) NIDDM – type II – Senile M.
- ❖ **Diabetic Retinopathy:** It is a bilateral retinal affection due to D M.

### Risk factors of DR:

- 1) **Duration: Most important factors.**
  - ❖ 50% will develop D. retinopathies after 10 years.
  - ❖ 90% will develop D. retinopathies after 30 years.
- 2) **Good metabolic control:** Will not prevent but will delay for few years.
- 3) **Miscellaneous factors:**
  - a) Pregnancy.
  - b) HTN.
  - c) Renal disease.
  - d) Smoking.
  - e) Anemia.

### Pathogenesis of DR:

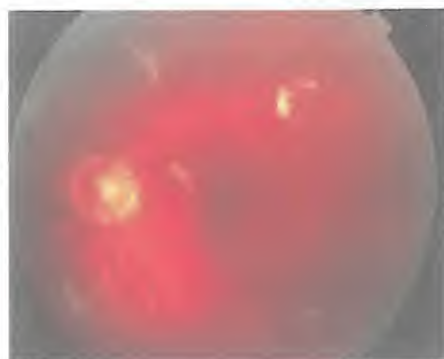
- 1) **Micro-vascular leakage:**
  - ❖ Progressive loss of pericytes resulting in dilated capillary segments or micro –aneurysms
    - **Break down of inner blood retinal barrier**
    - **Leakage:** Edema – Hard exudation – Hge (Dot Hge – Flame Hge).
- 2) **Micro -vascular occlusion: Why?**
  - a) **2 changes wall:** (Basement membrane thickening – Proliferation of endothelium).
  - b) **2 changes in blood constituent:** (Increase platelets slickness – Dec. RBCs deformability).
  - ❖ **Result:** The capillary occlusion results in retinal non perfusion → Retinal ischemia.
  - ❖ **To solve retinal ischemia following occurs:**
    - a) Arterio venous shunts (IRMA).
    - b) Neo-vessel formation (NVDs – NVEs).
  - **As a result of formation of vasogenic material.**

The difference between A/V shunts and neo-vessels is that A/V shunts lie within the retinal layer, but neo-vessels are present above retinal surface in the plane between the retina and the vitreous.

## Types of DR and its treatment:

### 1) Background DR:

- ❖ Micro aneurysms (detected by fluorescein angiography).
- ❖ Retinal Hge: Dot and flame Hge.
- ❖ Hard exudates.
- ❖ Retinal edema.
- ❖ Treatment: Medical:
  - Baby aspirin 75 mg/day.
  - Doxum 500 mg cap (↓ exudation).

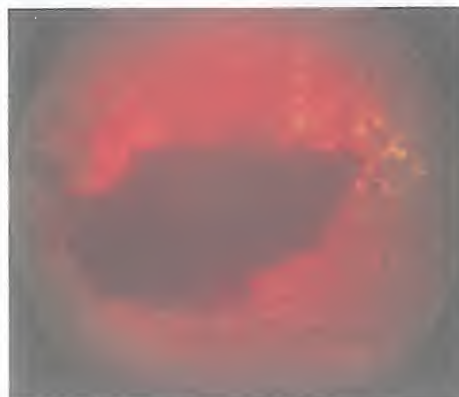


### 2) Pre-proliferative DR:

- ❖ Venous changes (Beaded).
- ❖ Cotton wool spots or soft exudates (micro -infarctions).
- ❖ Blot Hges (Hemorrhagic infarctions).
- ❖ IRMA or A/V shunts.
- ❖ Just follow up (close follow up).

### 3) Proliferative DR:

- ❖ Neo-vessel formation NVDs – NVEs. →
- ❖ Vitreous detachment (PVD).
- ❖ Vit. Hge and subhyaloid Hge.
- ❖ Treatment: Pan retinal argon laser photocoagulation.



Normally there is vitreous-retinal adhesions at

1) On schias

2) Around optic disc

3) At macula

4) Around B.V

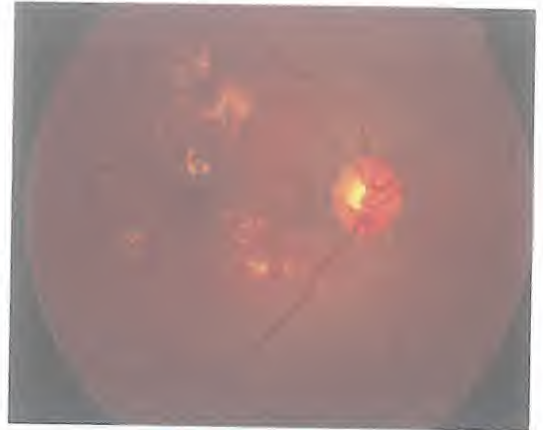
#### 4) Diabetic maculopathy:

Presence of any retinal thickening or hard exudates within 1500 Mm of the center of the fovea. If the edema or exudates are 500 Mm from the fovea, it is called clinically significant macular edema. Involvement of the fovea by edema and/or hard exudates is the most common cause of visual impairment in diabetic patients.

5) Treatment: (Focal Laser – Macular grid).

#### **Complications of DR:**

- ❖ Persistent vit. Hge → Vitrectomy
- ❖ Epi-retinal membrane → Vitrectomy
- ❖ Tractional R.D. → Vitrectomy
- ❖ Rubeosis iridis → Pan retinal photocoagulation
- ❖ Ischemic optic neuropathy.



## New classification of DR

### 1) Non - proliferative D.R.:

#### a) Mild to moderate:

- ❖ Microaneurysms.
- ❖ Dot hge in < 4 quadrants.
- ❖ Hard exudates.
- ❖ Retinal edema.

#### b) Moderate to severe:

- ❖ Retinal hge in 4 quadrants.
- ❖ Venous beading.
- ❖ Cotton wool spots.

#### c) Severe NPDR: anyone of:

- ❖ Severe ret. hge in 4 quadrants.
- ❖ Venous beading in 2 quadrants.
- ❖ IRMA in One quadrant.

### 2) Proliferative D.R.:

a) Early or mild: NVDs <  $\frac{1}{3}$  disk diameter.

b) High risk P.D.R.:

- ❖ NVDs >  $\frac{1}{3}$  disk diameter.
- ❖ NVEs >  $\frac{1}{2}$  disk diameter.
- ❖ Vitreous hge & epi-retinal hge.



## Ocular manifestations of D.M

- 1) **Lid:** Blepharitis, recurrent sty.
- 2) **Conjunctiva:** Conjunctivitis.
- 3) **Cornea:** Keratitis (ulcer).
- 4) **Ant. chamber:** Hyphema (from Rubeosis iridis).
- 5) **Iris:** Rubeosis iridis.
- 6) **Lens:** Complicated cataract.
- 7) **Changes in refraction:**
  - a) Hyperglycemia → **Index Myopia**.
  - b) Hypoglycemia → **Index Hypermetropia**.
- 8) **Vitreous:** Vitreous Hge (from neo-vessels in DR).
- 9) **Retina:**
  - a) DR.
  - b) CRVO → Retinal Hge.
  - c) **Lipemia retinalis:** Due to hyperlipidemia (showing pale fundus & milky vessels).
- 10) **IOP:** ↓ in diabetic coma due to Hypotony.
- 11) **Optic nerve:** Optic neuritis & Optic chiasma lesions.
- 12) **Orbital:** Orbital cellulitis + Endocrinal endophthalmia.
- 13) **Extra-ocular muscles:** Paralytic squint especially lateral rectus.
- 14) **Post-operative complications:** Hge, infection, delayed wound healing.

## Hypertensive Retinopathy

### Pathogenesis:

- ❖ Primary response of the retinal arterioles to hypertension is **vasoconstriction**  
→ (if V.D. is severe leads to retinal ischemia).
- ❖ If the elevated BP persists (sustained HTN) → **disruptions of blood retinal barrier** → **Leakage**.

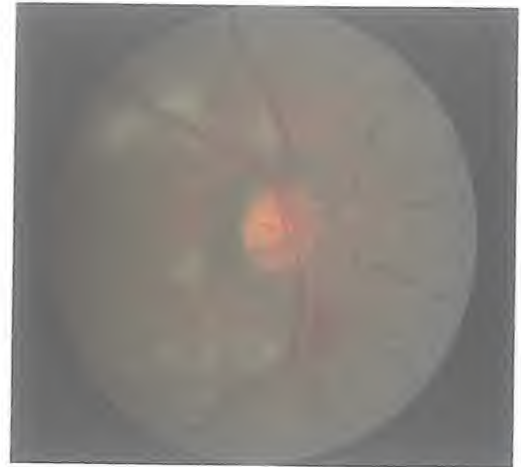
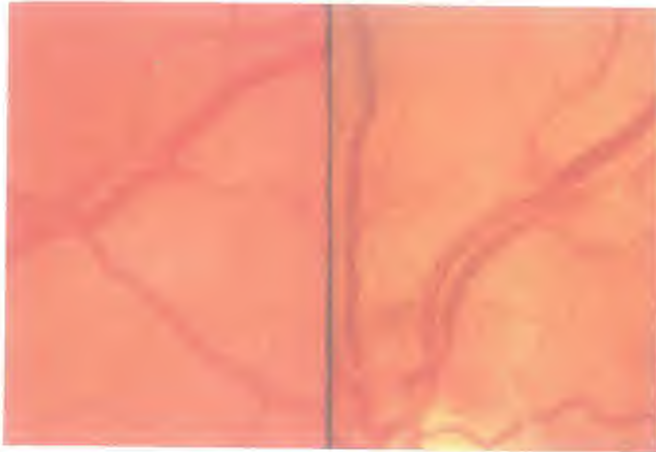
### Fundus pictures:

- 1) **Vasoconstriction of retinal arteries** which is either generalized or focal V.C.
- 2) **Cotton wool spots** (if V.C. is severe).
- 3) **Leakage in the form of:**
  - a) Retinal Hge (flame shaped Hge).
  - b) Retinal edema.
  - c) ONH edema.
  - d) **Hard exudates** deposited in the OPL (Henley's fiber layer) forming macular fan.

#### 4) Arterio Sclerosis:

##### a) Thickening of the vessel wall with changes at the A -V crossings:

- ❖ **Salus Sign:** Deflection of the veins at the A -V crossings.
- ❖ **Bonnet's sign:** Banking of blood inside the veins distal to A -V crossings.
- ❖ **Gun's sign:** Tapering of the veins on either side of A -V crossings.



##### b) Changes in light reflex: Copper wiring – Silver wiring.

##### c) Grading of Hypertensive retinopathy:

- ❖ **Grade 1:** Mild generalized V.C.
- ❖ **Grade 2:** Severe V.C + Salus sign.
- ❖ **Grade 3:** Cotton wool spots + Hard exudates + Hge + Ret. edema + Bonnet sign + Gun's sign + Copper wiring. (All except Disc edema and silver wirings).
- ❖ **Grade 4:** Disc edema and silver wiring + all the above.

#### **Other ocular manifestations of Hypertension:**

Hypertension also plays a role in **branch retinal vein occlusion, retinal artery occlusion, ischemic optic neuropathy, and ocular motor nerve palsy**. Uncontrolled hypertension also has an adverse effect on diabetic retinopathy

## Retinopathies Of Malignant Hypertension And Renal Failure

Simply it is grade 4 hypertensive retinopathy (all the hypertensive changes),

It develops on top of benign HTN but if develop rapidly; the vessels are attenuated and straight.

# Retinopathy of toxemia of pregnancy

**Definition** Bilateral retinal affection due to hypertension as a result of pregnancy (Toxemia of pregnancy or pre-eclampsia).

**Clinical Picture:** Onset: Affects females after 20<sup>th</sup> week of pregnancy.

## Symptoms:

- 1) Mild cases: Asymptomatic.
- 2) Severe cases:
  - a) **Metamorphopsia** (distorted objects) (Due to macular edema).
  - b) **Sudden painless ↓ V/A** if exudative R.D occurs.

## Signs:

- 1) VC of retinal arterioles.
- 2) Cotton wool spots.
- 3) Ret. Edema – Hge – Hard exudation
- 4) Disc edema.

## **Complications:**

- ❖ Macular edema.
- ❖ Exudative R.D, which when occur pregnancy should be terminated.

# Photoreceptor Dystrophies

## Retinitis pigmentosa

**Definition:** Group of hereditary disorders characterized by progressive loss of photoreceptors and RPF functions.

- ❖ There are many variants of the disease, Typically, there is a diffuse bilateral, symmetrical retinal dystrophy affecting mostly rod functions

## **Etiology:**

### 1) Unknown:

- ❖ Abiotrophy (cong. weakness of tissues).
- ❖ Photo -toxicity.
- ❖ Hereditary plays a role.

### 2) Inheritance:

- ❖ Autosomal dominant (most common).
- ❖ Autosomal recessive.
- ❖ X-linked.
- ❖ Sporadic.





### **Pathology:**

- ❖ Arteriolar narrowing.
- ❖ Degeneration starts at equator (blood supply is less than at the center of the retina).
- ❖ Coarse pigmentary change with a pen-vascular arrangement (spider pigmentation) due to migration of RPE in the inner retinal layers.
- ❖ Then the degeneration and pigmentary changes spread peripherally and centrally.
- ❖ The central vision become affected when macula is involved (Maculopathy).

### **Clinical Picture:**

#### **Symptoms:**

- 1) Defective night vision (Night blindness).
- 2) Later progressive loss of vision.

#### **Signs:**

##### 1) Typical R.P:



+ Field changes: Early annular → Late tubular field.

[(50°) around center of fixation]

##### 2) Atypical R.P:

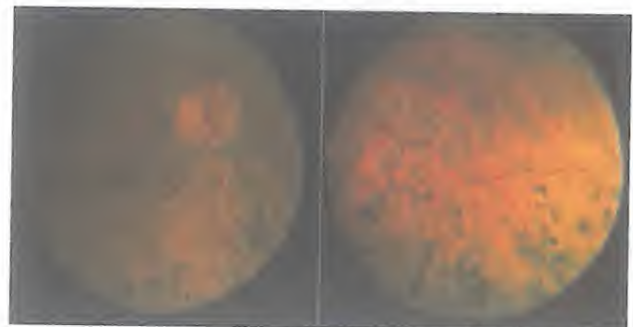
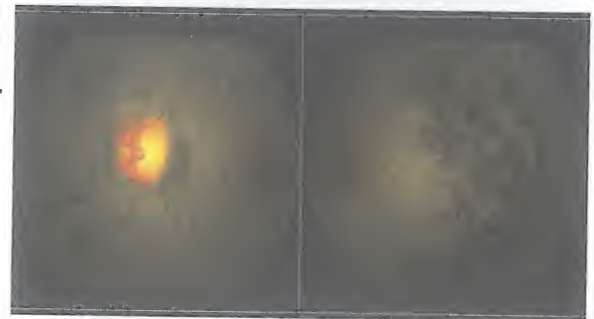
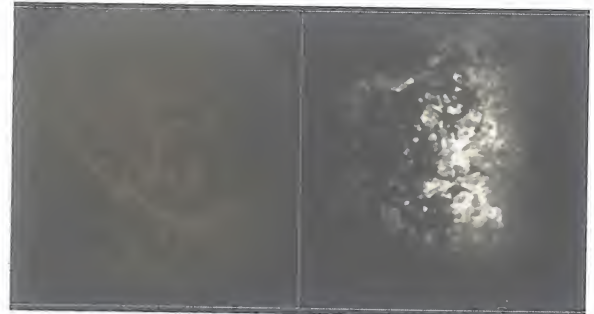
- a) R.P sin pigment.
- b) Sectorial R.P.
- c) Pen-centric R.P.

### **Common Associations:**

- a) Complicated cataract.
- b) Primary open angle glaucoma.
- c) Keratoconus.
- d) Myopia.
- e) Vitreous changes (PVD).

### **Investigations: (Electrophysiological tests):**

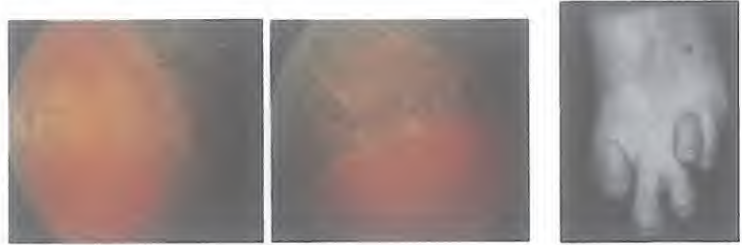
ERG: (Electro-retinal gram): Reduced amplitude initially of scotopic laser of photobic wave.



## Syndromes associated with R.P:

1) **Refsum's disease:** Defective metabolism of phytanic acid which infiltrates many tissues.

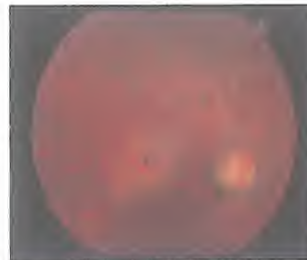
- ❖ Deafness
- ❖ Ataxia
- ❖ Polyneuropathy
- ❖ Cardiac arrhythmia
- ❖ **Salt and pepper Ret. Pigment.**



2) **Bardet-biedl syndrome:** Prognosis: Loss of VA due to: Maculopathy, Consecutive OA &

Complicated cat.

- ❖ Obesity
- ❖ Hypogonadism
- ❖ Mental retardation
- ❖ Polydactyl
- ❖ **+ Bull's eye Maculopathy**



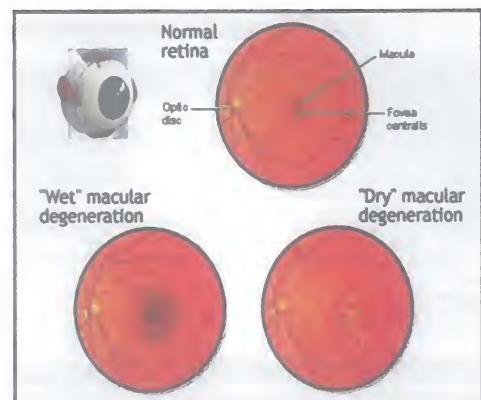
## Age-related macular Degeneration (AMD)

❖ It is the leading cause of irreversible severe visual loss above 60 years.

### ❖ **Two types of AMD:**

#### 1) **Dry AMD (non-exudative type):**

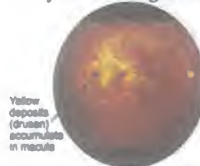
- a) Accounts for 90% of cases of AMD.
- b) Results from: slowly progressive atrophy of RPE and photo receptors
- c) Clinical Picture: → Focal hyperpigmentation.  
→ Circular areas of RPE atrophy through which large choroidal vessels are seen.



#### 2) **Wet type of AMD:**

- a) Rapid ↓ in VA + Metamorphopsia.
- b) Signs: (choroid neovascular membrane).
- c) Sub-retinal exudation of Hge and fibrosis.

Dry Macular Degeneration



Wet Macular Degeneration



### ❖ **Treatment:**

- a) **Laser photo coagulation.**
- b) **Photo dynamic therapy (PDT):** A dye is injected intravenous and the lesion is exposed to a laser of a certain wavelength to produce a chemical reaction and coagulate the neo-vessels.
- c) **Intra-vitreous injection of anti-VEGFs.**

## Anti Vascular Endothelial Growth Factors (Anti-VEGFs)

- ❖ **Responsible for:** Growth of new blood vessels by stimulating the endothelial cells, which form the walls of the vessels and transport nutrients and oxygen to the tissues.
- ❖ **Neo-vascularization:** When the retina begins to wither from lack of nutrition (Ischemia), the VEGF goes into action to create new vessels.
  - It acts as: restorative function in other parts of the body.
- ❖ **In the retina:** However, vessels do not form properly and leaking results.
  - This leakage: causes scarring in the macula & eventual loss of central vision.
- ❖ **Mechanism of action of anti-VEGFs:**

They prevent VEGF from binding with the receptors on the surface of the endothelial cells. In most cases the drugs are injected into the vitreous of the eye ball.
- ❖ **Available Anti-VEGFs in ophthalmology:**

Avastin (Bevacizumab) – Lucentis (Ranibizumab) – Macugen (Pegaptanib sodium)
- ❖ **Uses of Anti-VEGFs:**

PDR – Diabetic macular edema – CRVO – Branch retinal vein occlusion with macular edema – Wet AMD

## Retinal detachment

**Definition:** It is a condition in which retina is separated into 2 layers, inner a sensory retina in one side and the RPE in the other side.

### Types:

- 1) **Primary R.D.** (Simple R.D. - Rhegmatogenous R.D)
- 2) **Secondary R.D.**

## Primary R.D.(Rhegmatogenous R.D)

**Definition:** Formation of retinal break through which liquefied vitreous enters between retinal layers and causes retinal detachment or separation.

**N.B:** The liquefied vitreous in the sub-retinal space is known as sub-retinal fluid.

### Risk Factors:

- 1) High myopia.
- 2) Trauma.
- 3) Aphakia.
- 4) Family history of R.D.
- 5) R.D in the other eye.



## Retinal breaks:

### 1) Types:

- ❖ Tear (dynamic vitreo-retinal traction).
- ❖ Hole (chronic atrophy and sensory retina).
- ❖ Most common site of a hole formation is the fovea. (Why??)

### 2) Morphology of breaks:

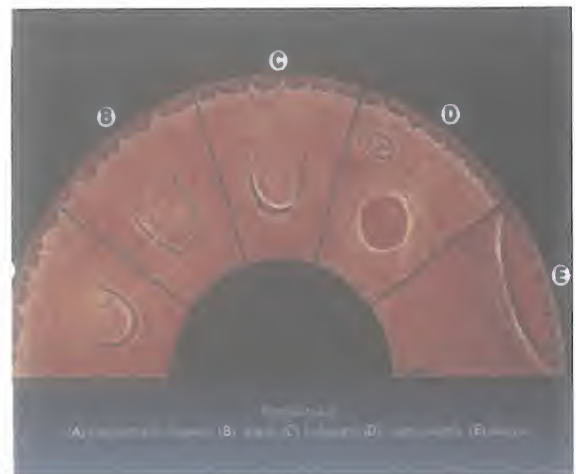
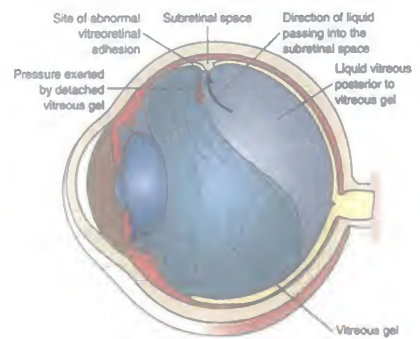
a) **Hole:** Rounded or oval (1)

b) **Tears:**

- ❖ Horse shoe (2) – Incomplete horse shoe (3) – Arrow head (4)
- ❖ **Operculated tears:** In which the flap is completely torn away from the retina.
- ❖ **Giant tear** (more than 90° of circumference of the globe).
- ❖ **Dialysis or Retinal disinsertion.**

c) **Site of the breaks:**

- ❖ Oral breaks. (At ora serrata or vitreous base).
- ❖ Post oral breaks (between ora and equator).
- ❖ Equatorial breaks.
- ❖ Post equatorial breaks.
- ❖ Macular breaks.



## Clinical picture:

### Symptoms:

#### Early:

- ❖ **Photopsia (flashes of light)** due to sudden traction of vitreous on retina.
- ❖ **Floater: (Musca volitans):** Due to:
  - Sudden shower of minute red colored or dark spots indicating vit. Hge if tearing of retinal vessel occurred.
  - Operculum of retinal tear floating within the vitreous casting shadow on the retina.

#### N.S.:

Photopsia may occur during PVD without associated retinal tear.

Floater may occur as a result of vit. degeneration in cases of high myopia or old age.

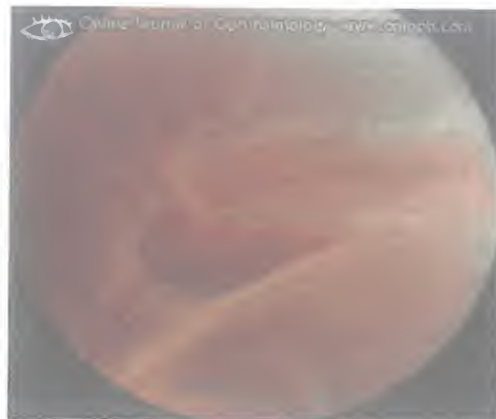
#### Late:

- ❖ **Rapid visual field defect** perceived by the patient as a (black curtain).
- ❖ **Loss of central vision** if macula is involved.

## Signs:

### 1) Anterior segment signs:

- a) **RAPD** if there is extensive R.D.
- b) **Decrease in the IOP** by about 5 mmHg.
- c) **Mild Iritis:** Flare – Cells.
- d) Anterior vitreous face shows **tobacco dust**.
- e) Red reflex: **Greyish reflex**.



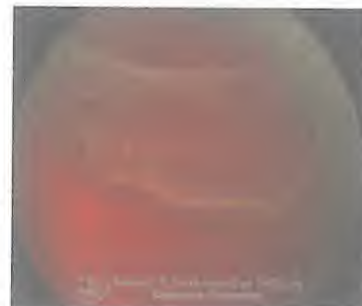
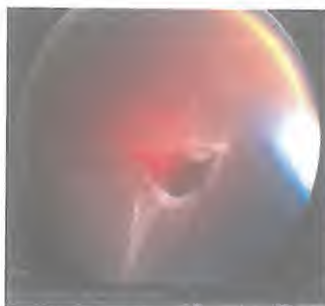
### 2) Post segment signs (fundus examination):

a) Site of the break appears as: A red discontinuity within the greyish reflex.

b) The detached retina may show:

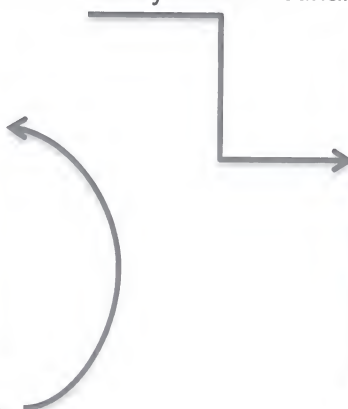
#### ❖ Fresh Ret. Detachment:

- Convex configuration.
- Greyish in color.
- Thickened due to edema.
- Moves freely with eye movements.



#### ❖ Long standing R.D:

- Thinned retina (atrophic).
- Sub-retinal demarcation line or high water mark (its presence means that detachment has occurred from at least 3 months secondary to intra-retinal cysts {from one year}).



#### ❖ Proliferative vitreoretinopathy:

- **It is caused by:** proliferation of membranes on the inner retinal surface (epi-retinal) or sub-hyaloid or sub-retinal.
- **Clinically:** it appears as fixed retinal folds.
- **Significance:** in order to repair detached retina, you must remove these membranes or otherwise failure of surgery is common.

## Examination of a case of R.D.:

### 1) Indirect ophthalmoscope:

- a) Extent of RD.
- b) Site, shape, size, and number of retinal breaks.
- c) State of the macula.
- d) Presence & severity of PVR.

### 2) Slit-lamp Bio-microscopy:

- a) Using **Goldmann three-mirror contact lens**.
- b) Using a **non-contact lens +90 or +60 diopters lens**.

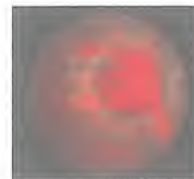
### 3) Ultra-sonography:

- a) **Useful in:** Patients with opaque media, suspected to have RD.
- b) **Exudative RD** with suspected intraocular tumor.

## Treatment of 1<sup>ry</sup> R.D.:

### 1) Prophylactic treatment:

- ❖ If there is retinal break but **vitreous do not yet enters to cause R.D.**
- ❖ **Aim of treatment:** Is to induce sterile Chorio-retinitis leading to adhesions between choroid and retina around the break preventing vitreous entrance.
- ❖ **Method:**
  - a) Cryo applications: single row of Cryo applications around break.
  - b) Argon laser photocoagulation: doing 2 rows of laser burns around break.



### 2) Actual treatment of RD:

#### a) Scleral buckling or conventional RD surgery or standard R.D. Surgery:

##### ✦ Steps:

- ❖ To close the break by **Cryo applications**.
- ❖ To **estimate SRF** either being absorbed by the underlying choroid or needs artificial drainage by doing surgical sclerotomy.

**N.B.:** Signs indicating that SRF will not be absorbed by the choroid are:

1) Large amount of SRF

2) High viscosity of SRF (Long standing)

3) Atrophic choroid (High myopia)

- ❖ Creating of inward indentation of sclera using scleral explants at the site of the break either using → tire and band or radial.



b) **Pars plana vitrectomy:** It is an intraocular surgery in which the vitreous gel is removed first to gain access to the retina in order to repair the R.D.

✚ **Steps:** 3 pars plan incisions at:

- ❖ 10° Clock (vitrectomy probe).
- ❖ 1° Clock (illumination source).
- ❖ 5° Clock (infusion canula).

1) **Closure of the break using endo-laser.**

2) **Drainage of SRF** if needed by doing retinotomy.

3) **Replacement of vitreous by silicone oil** to push the retina towards the choroid.

✚ **Indications :**

- 1) Break: → Site (posterior), Number (multiple) & Size (giant).
- 2) Vitreous opacities.
- 3) PVR.

**W.H. : Silicone oil should be removed after 6 months. It may cause complications.**

→ Silicon cataract

→ Emulsification

→ 2° glaucoma

→ Inverted hypopyon

✚ **Other Indications of vitrectomy in case of proliferative diabetic retinopathy:**

- 1) Unresolved vitreous hge.
- 2) Tractional RD involving the macula.
- 3) Combined tractional and Rhegmatogenous RD.

## 2<sup>ry</sup> R.D

### 1) Tractional R.D:

**Definition:** R.D due pulling on the retina by contractive vitreous membranes.

**Causes:**

- 1) P.D.R.
- 2) PSR (Proliferative sickle cell retinopathy).
- 3) Retinopathy of prematurity.
- 4) Penetrating posterior segment trauma.
- 5) Cyclitic membrane: in case of iridocyclitis.

**Clinical picture:**

**Symptoms:**

- ❖ No Photopsia (traction is gradual).
- ❖ No floaters (vitreous is opaque).
- ❖ Field defect is very gradually progressive.

**Signs: Detached retina:**

- ❖ Has concave contour.
- ❖ Highest elevation at site of traction.
- ❖ Mobility of retina is severely restricted.
- ❖ SRF is absent or minimal of unknown origin.

### 2) Exudative R.D:

**Definition:** Retina is pushed by the exudating fluid from choroid.

**Causes:**

- 1) **Systemic causes:** as severe HTN, toxemia of pregnancy and hypo-proteinemia.
- 2) **Local causes:**
  - a) Tumors (choroidal tumors).
  - b) Inflammations (Harada syndrome).
  - c) Choroidal neo-vascular membrane.

**Clinical picture:**

**Symptoms:** No Photopsia – No floaters (except if there is vitritis) – Rapid field loss.

**Signs: Retina:**

- ❖ Convex contour.
- ❖ Smooth surface.
- ❖ Shifting fluid sign.
- ❖ No retinal break

# Retinopathy of prematurity

## Definition & Pathogenesis:

- ❖ **Retinopathy of prematurity (ROP)**: is a proliferative retinopathy affecting preterm infants of low birth weight who have been **exposed to: high ambient oxygen concentrations**.
- ❖ The retina is unique among tissues in that it has **no blood vessels until fourth month of gestation**, at that time **blood vessels arise from the hyaloid artery towards the retinal periphery**.
- ❖ The vessels reach **the nasal retinal periphery by the 8<sup>th</sup> month of gestation**, and the **temporal retinal vessels reach the temporal retinal periphery 1 month after delivery**.
- ❖ **So, in preterm infants**, the incompletely vascularized temporal retina is source of trouble being ischemic.
- ❖ It has been postulated that **exposure to oxygen free radicals inhibit spindle cell migration** which is needed for normal blood vessels growth and the ischemic temporal retina in turns produces angiogenesis factors responsible for development of ROP.
- ❖ Because of the sequential nature of progression of ROP, the standards of practice now demand **carefully timed retinal examinations of at-risk infants to minimize the risk of visual loss**.

## Screening:

- ❖ **Infants with a birth weight of < 1500 gm or with a gestational age of < 32 weeks** should have dilated Fundus examinations every **2 weeks till 36 weeks** with the first examination should normally be performed at the **31<sup>st</sup> to 33<sup>rd</sup> week of post-conceptional**.

## Treatment:

- 1) Should generally be accomplished **within 72 hours of examination**, if it determined the presence of threshold ROP.
- 2) Studies have shown that treatment with **cryotherapy or laser therapy** is associated with a **40% decrease in the occurrence of posterior retinal tractional folds or detachments** and a **25% decrease in the incidence of blindness when evaluated 5 years later**.



# The Vitreous

- ❖ The vitreous humor constitutes **two-thirds of the volume of the entire globe**.
- ❖ It is a **gel-like substance** composed of **more than 99% water**. The remaining part is formed of **collagen and hyaluronic acid**, giving the vitreous its **rigidity and viscosity (jelly like)**
- ❖ The vitreous is bounded **anteriorly by the lens, iris and ciliary body**, and **posteriorly by the retina and optic disc**.
- ❖ **The outer portion of the vitreous body** is denser than the center and called **the cortex**, and its surface is called **the hyaloid membrane**.
- ❖ In youth, **the anterior hyaloid** is fixed firmly to the posterior lens surface; this attachment becomes weaker with age.
- ❖ **Cloquet's canal** runs **anteroposteriorly in the center of the vitreous** and is the site of **the embryonic hyaloid artery**. The strongest attachment of the vitreous is to **the retina and pars plana** in the area of the vitreous base, straddling the ora serrata

## ❖ **Functions of the vitreous:**

- 1) Stabilizes the volume of the globe
- 2) Acts as a cushion for the retina
- 3) One of the optical media of the eye.

## ➤ **Aging changes of the vitreous:**

- **Age-related changes of the vitreous include liquefaction (synchysis):** This results in the formation of fluid lacunae inside the vitreous gel. These may rupture through the cortical vitreous and cause separation of the cortical vitreous from the inner retinal surface (posterior vitreous detachment).
- The patient may complain of **flashes of light (photopsia)** and **vitreous floaters**.
- The patient describes it as black dots, rings or other shapes moving in the field of vision (**musca volitantes**)

## ➤ **Causes of Vitreous Hemorrhage:**

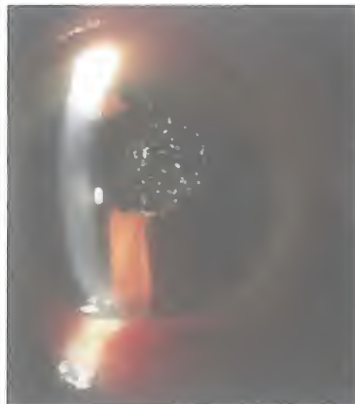
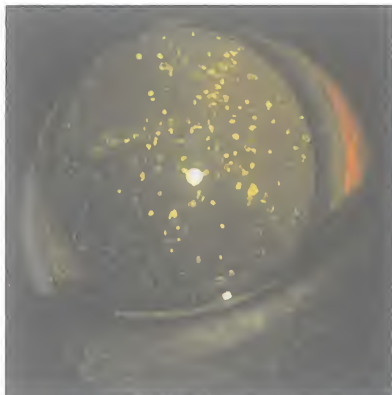
- 1) Proliferative retinopathies, as diabetic retinopathy.
- 2) Retinal breaks.
- 3) Central retinal vein occlusion.
- 4) Trauma.
- 5) Blood diseases as anemia, leukemia and purpura.
- 6) Intraocular tumors.

➤ **Fate of vitreous hemorrhage:**

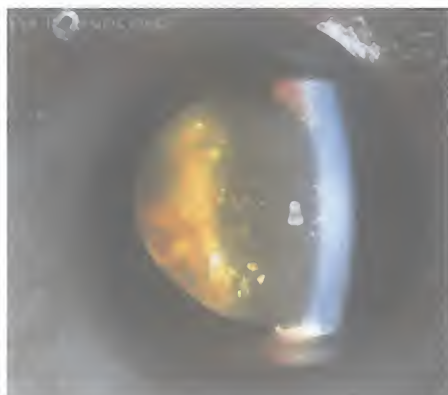
- Absorption is very slow.
- Retinitis proliferans leading to retinal detachment.

➤ **Degenerative vitreous changes:**

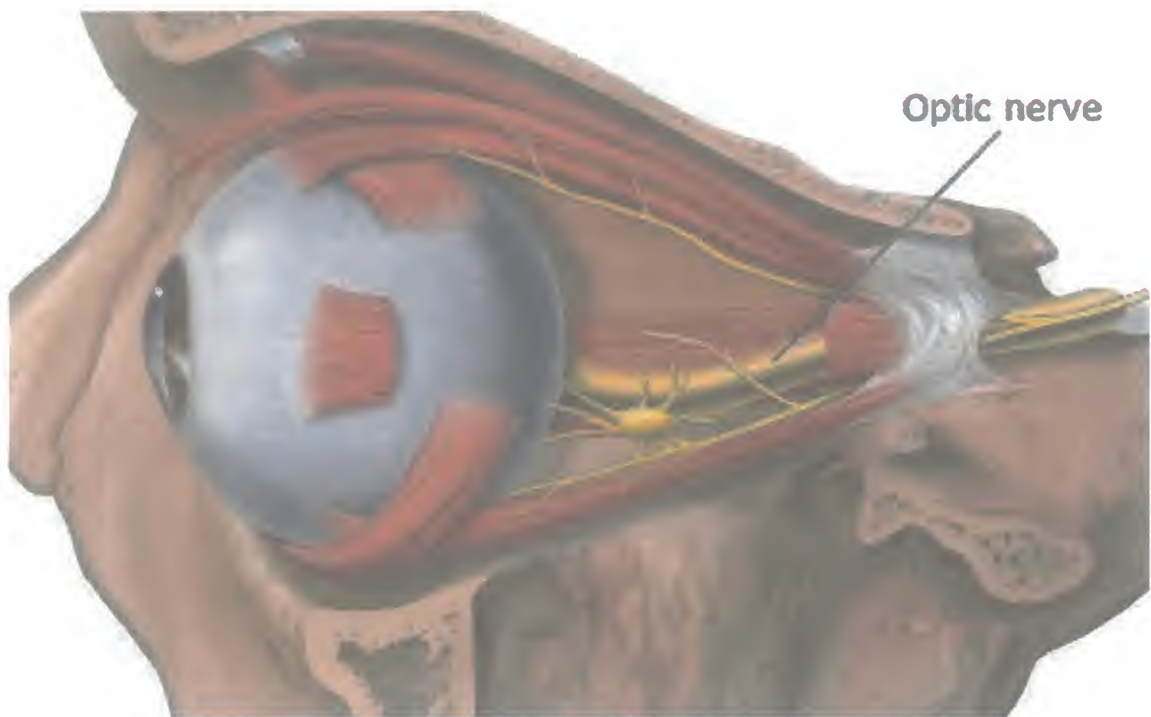
- 1) **Asteroid hyalosis:** The condition is characterized by the presence of small oval, reflective, white-to-yellow opacities attached to formed vitreous.
  - Usually unilateral in old age. Patients are usually asymptomatic and vision is rarely affected.
  - The opacities are formed of calcium and phosphorus.



- 2) **Synchysis Scintillans:** This condition is characterized by the presence of fine, highly refractive crystals in degenerated (liquified) vitreous, usually in young age.
  - These crystals are composed of cholesterol esters.
  - The condition is usually bilateral.
  - The crystals are mobile, settle down by rest, and disperse on eye movement.



# Optic nerve





# Anatomy

**Optic nerve head:** Is the optic disc (papilla), which is seen with ophthalmoscope.

**Optic nerve:** It runs from lamina cribrosa to optic chiasma:

1) About 5 cm:

a) Intra-ocular part: 0.6 mm.

c) Intra-canalicular part: 6 mm.

b) Intra-orbital part: 30 mm.

d) Intra-cranial part: 10 mm.

2) Only myelinated up to lamina cribrosa.

3) About one million fibers.

4) These fibers have **no neurilemma**, thus no regeneration of the injured or destroyed fibers occurs.

## Diseases Of The Optic Nerve

### 1) Papilledema

**Definition:** It is passive (non-inflammatory) edema (transudate mostly water) of optic Disc usually due to increase of ICT.

- ❖ Passive = congestion = venous = non-inflammatory.
- ❖ Active = arteriolar dilatation = inflammation = papillitis.

**Mechanism (of papilledema with + ICT):**

1) **(Mechanical) theory:** Intra-cranial subarachnoid space is communicating with subarachnoid space around optic nerve. So any increase in ICT will be transmitted to Optic nerve, pressing on CRV → Transudation.

2) **Recently:** Papilledema is due to **block of the axoplasmic flow** due to ↑ ICT in the subarachnoid space around the optic nerve

Later: the swollen axons compress the capillaries & veins → Congestion → Transudation.

**Etiology:**

1) **Intra-cranial causes:** Due to ↑ IC pressure e.g. in:

➤ **Neoplasm:** 70% of cases of papilledema are brain tumors:

- ❖ Always **bilateral** unless one nerve is atrophic.
- ❖ In front lobe tumors, one disc may be directly compressed by the tumor leading to 1<sup>ry</sup> optic atrophy, while the other disc becomes swollen. This is known as **Foster-Kennedy Syndrome**.

- **Inflammation** (Brain Abscess, tuberculoma & meningitis).
- **Vascular:** Sub-arachnoid Hge & cavernous sinus thrombosis.
- **Pseudo tumor cerebri:**
  - ❖ Benign increased ICT with no intracranial lesions or mass.
  - ❖ More in young females due to: Hypervitaminosis A and D, oral contraceptives & endocrine disorders (Addison's disease & Cushing's syndrome).

## 2) Other causes:

### a) Orbital (rare): Due to:

- ❖ Retro-bulbar mass as: Tumors or cysts.
- ❖ Inflammation (orbital cellulitis).

### b) Ocular: Due to:

- ❖ CRVO.
- ❖ Hypotony (V.D - Transudation).

### c) Systemic: Due to:

- ❖ Malignant hypertension.
- ❖ Toxemia of pregnancy (Eclampsia).
- ❖ Anemia & Polycythemia.
- ❖ Renal retinopathy.

## N.B 1: Sign

1) **Bilateral Papilledema:** in intra-cranial causes & systemic.

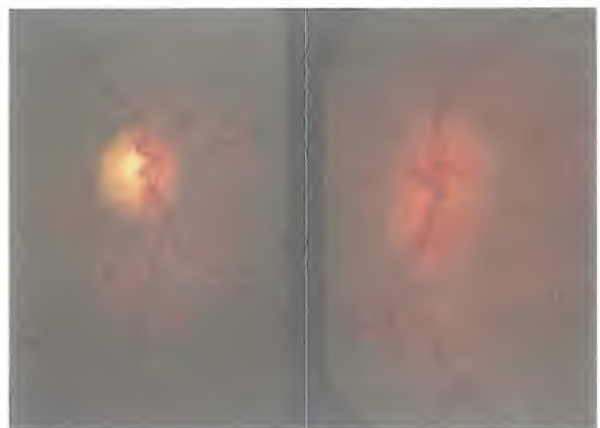
2) **Unilateral Papilledema:** in orbital causes, ocular causes & Foster -Kennedy syndrome.

## N.B 2

1) **Papilledema:** takes lto 5 days to appear after rise of ICT.

2) **Foster -Kennedy syndrome:** in frontal lobe tumors with:

- Optic atrophy on the same side: Due to pressure effect.
- Papilledema on the other side occurs later: Due to ↑ ICT (as atrophic optic nerve not capable of being edematous).



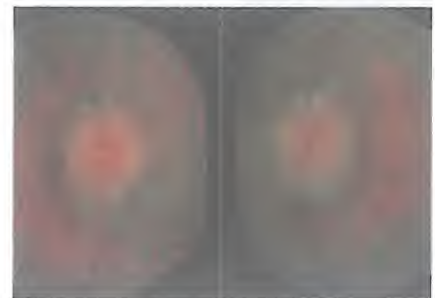
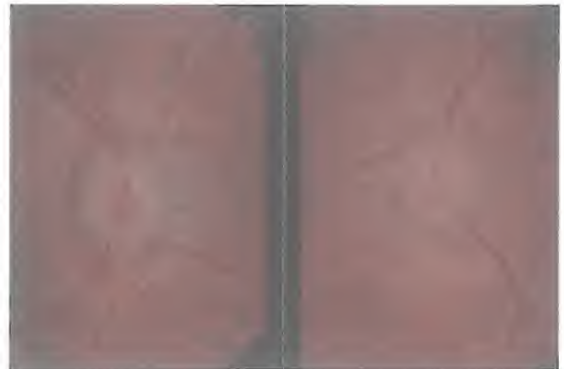
## Clinical picture:

### Symptoms:

- 1) **Symptoms of ↑ ICT:** headache, blurring, projectile vomiting (not preceded by nausea), Diplopia due to 6<sup>th</sup> nerve palsy
- 2) **Visual symptoms:**
  - a) **Early:** normal (sometimes Amaurosis Fugax).
  - b) **Late:** ↓ gradually up to loss → (due to optic atrophy due to pressure & ischemia).

### Signs:

- 1) **Pupil:**
  - a) **Early:** normal.
  - b) **Late: with occurrence of optic atrophy:**
    - ❖ If bilateral → Dilated irreactive pupil
    - ❖ If unilateral → RAPD
- 2) **Fundus: Like CRVO:**
  - a) **Retinal:** Hge & edema.
  - b) **Macula:** exudates (fan).
  - c) **Veins:** engorged, tortuous and absent venous pulsations
  - d) **Disc: (pseudo-papillitis):**
    - ❖ Filling of cup.
    - ❖ Elevated (up to 9D = 3mm).
    - ❖ Ill-defined edges.
    - ❖ Hyperemic.
- 3) **Field:**
  - a) **Early:** enlargement of the blindspot and central scotoma for blue.
  - b) **Late:** Contraction of field due to atrophy



## Investigation:

- 1) **Field of vision.**
- 2) **Color vision:** Central scotoma for blue.
- 3) **Radiology: X-ray, CT- scan and MRI brain**
- 4) **VEP:**
  - a) **Early:** normal.
  - b) **Late:** affected.

**Complications:** Post - papilledemic (2<sup>ry</sup>) optic atrophy.



**Prognosis:** Bad in marked edema (if more than 5 D).

**D.D.** Papillitis – Pseudo papillitis

### **Ocular manifestations of brain tumors:**

#### **1) Frontal lobe:**

- a) Foster-Kennedy Syndrome.
- b) Chiasmal lesions.
- c) Orbital apex syndrome.
- d) Loss of voluntary gaze.

#### **2) Temporal lobe:**

- a) Papilledema.
- b) Formed visual hallucinations.
- c) Superior homonymous quadrantanopia.

#### **3) Parietal lobe:**

- a) Papilledema.
- b) Saccadic pursuit.
- c) Superior homonymous quadrantanopia.

#### **4) Occipital lobe:**

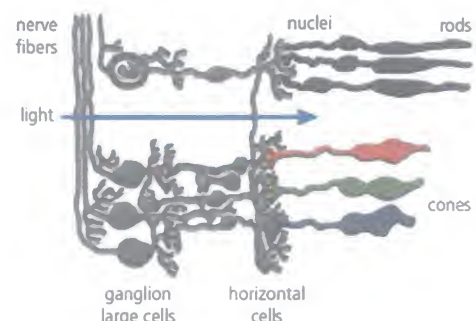
- a) Papilledema.
- b) Simple visual hallucinations.
- c) Blindness with normal pupils and ERG (cortical blindness).
- d) Congruent hemianopia with macular sparing.

### **Treatment:**

- 1) Treatment of the cause.
- 2) Dehydrating agents & decompression of optic nerve sheath.

### **Types of cones responsible for color vision:**

- 1) **B**: sensitive for blue & affected by ischemia or pressure as they are crowded.
- 2) **R**: sensitive for red affected by inflammations & toxins.
- 3) **G**: sensitive for green affected by inflammations & toxins.



## 2) Optic Neuritis

**Definition:** it is inflammation of the optic nerve.

**Classification:** it is classified into:

- 1) **Papillitis:** acute inflammation of the disc.
- 2) **Retro-bulbar neuritis:** inflammation of nerve behind globe (orbital part).

### Papillitis

**Definition:** Acute inflammation of the optic disc.

**Etiology: DIMA**

1) **Demyelinating disease** (M.S. is the commonest cause).

2) **Infection:**

- a) **1<sup>ry</sup>:** viral (polio - herpes) & bacterial (T.B. or \$).
- b) **2<sup>ry</sup>:** extension of infection from:
  - ❖ CNS (meningitis).                      ❖ Orbit (cellulitis).
  - ❖ Ocular: retinitis & uveitis.

3) **Metabolic diseases** e.g. DM & Vit B deficiency.

4) **Antigen antibody reaction** to a septic focus.

**Clinical picture:**

**Symptoms:**

- ❖ Rapid marked ↓ of vision up to HM.

**Signs:**

- ❖ **Fundus:** Disc is elevated 3D & retina less congested than papilledema.
- ❖ **Field:** Central scotoma for green & red.
- ❖ **Pupil:** Reactive unsustained reaction.

**Investigations:** **VEP:** Long latency and diminished amplitude

**Fate:**

- ❖ **Recovery (in most cases):** spontaneous.
- ❖ **Recurrence after recovery:** common.
- ❖ **Post papillitic (2ry) optic atrophy.**

**Treatment:** Treatment of cause + Systemic cortisone + Vit. B complex.



### D.D.: of papilledema & papillitis:

	Papilledema	Papillitis	Pseudo-papillitis
Pupil	Reactive (early)	Reactive (unsustained)	Reactive
I.C.T.	Present	Absent	Absent
Side	Commonly bilateral	Commonly Unilateral	Unilat. or bilat.
Mechanism	Passive process	Active process	Hypermetropes
Field	Central scotoma for blue	Central scotoma for green & red	Normal
Fundus	Disc is elevated Up to +9 D	Less elevation (About +3 D)	Less (about +2 D)
Vitreous	Clear	(inflammatory Cells)	Clear
Vision	Normal (early)	Markedly diminished	Hypermetropes

**N.B.: Pseudo-papillitis:** In high Hypermetropia due to crowding of nerve fibers of optic nerve due to small lamina cribrosa.

## Retrobulbar Neuritis

**Etiology:** as papillitis

**Clinical Picture:**

**Symptoms & signs:**

- 1) As papillitis + **painful eye movement** esp. up (SR muscle takes insertion from op. n sheath).
- 2) **Fundus:** normal disc & clear vitreous.

**Treatment:** as papillitis.

**N.B.:** Patient see nothing, doctor see nothing in:

- 1) Retro-bulbar neuritis.
- 2) Hysterical blindness.
- 3) Occipital cortex lesions.

**Toxic amblyopia:**

**Definition:** It is optic nerve damage due to exogenous toxins.

**Classification:**

- 1) Toxins which cause **central scotoma** as tobacco & ethyl alcohol.
- 2) Toxins which cause **contracted field** as quinine & salicylates.
- 3) Toxins which cause **severe optic atrophy** as methyl alcohol & arsenic.





### **Tobacco amblyopia:**

**It is due to:** degeneration of the ganglion cells of the papillo-macular bundle by the decomposition products of:

- 1) Nicotine.
- 2) Cyanide in tobacco smoke (smoking pipe and cigar) associated with vitamin B<sub>12</sub> ↓ as Vit B<sub>12</sub> normally neutralizes the effect of cyanide.

**Clinical Picture: Fundus:** normal.

**Field:** Centro-cecal scotoma for red & green (connect blind spot & fixation point).

### **Quinine amblyopia:**

**It is due to:** idiosyncrasy to the drug.

**Clinical Picture: Fundus:** cherry red spot (arterial spasm). **Filed:** tubular field (night blindness).

**Explanation:**

- a) Severe vaso-spasm → Close the peripheral vessels of the retina → Ischemia → Tubular field.
- b) **In severe cases:** there may be closed C.R.A → Cherry red spot.

### **Methyl alcohol amblyopia: methanol**

It is breakdown of methyl alcohol → Formaldehyde + Formic acid → Acidosis → Anoxia.

**Clinical Picture:**

- 1) Coma & death.
- 2) Degeneration of ganglion cells → Optic atrophy.

### **Treatment:**

- ❖ Stop the drug.
- ❖ Vasodilators.
- ❖ Vit. B complex.
- ❖ **In methyl alcohol Amblyopic:**
  - 1) Do gastric lavage with ethyl alcohol which makes competitive inhibition in liver with methanol.
  - 2) NaHCO<sub>3</sub> injection for acidosis.

## Anterior ischemic optic neuropathy

**Definition:** Partial or total infarction affecting the optic nerve due to: occlusion of the short post. Ciliary Arteries.

## a) Non-arteritic anterior ischemic optic neuropathy

- ❖ It occurs commonly in patients between 45 & 60 years, who are hypertensive, smokers or have high serum lipids.

**Symptoms:** An acute onset of painless, moderate to severe visual loss.

**Signs:**

**Color vision:**

It is impaired, in proportion to the visual acuity in contrast to optic neuritis, in which the color vision is markedly impaired out of proportion to the visual acuity.

**Fundus examination:**

Fundus examination shows **edema of the optic disc** that may be diffuse or affecting one sector.

The disc is surrounded by **splinter shaped hemorrhages**.

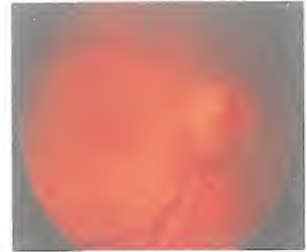
**Investigations:**

1) **Field vision:** Field defect in the form of altitudinal scotoma, arcuate scotoma & occasionally central if papillo-macular bundle is affected.

2) **Blood picture & serum lipids.**

**N.B.:** It is very important to exclude the arteritic type (see later).

**Fate:** Gradual resolution followed by optic nerve pallor occurs in most patients.



## b) Arteritic anterior ischemic optic neuropathy

- ❖ It is a serious type affecting mainly old patients above 70 years suffering from giant cell arteritis.

**Giant cell arteritis:**

- ❖ **Idiopathic vasculitis** affecting the media & adventitia of the large & medium sized arteries ending in the vascular occlusion.
- ❖ The arteries commonly affected are the **superficial temporal, vertebral, ophthalmic & short posterior ciliary arteries**.

**Symptoms:**

- ❖ Acute onset of **sudden severe visual loss** (up to no PL) & **periocular pain**.
- ❖ The affection may be bilateral, but simultaneous involvement is rare.
- ❖ **Constitutional manifestation:** in the form of **headache, fever, anorexia, weight loss & muscle pain** are common associations.

### Signs:

- ❖ Pale, swollen optic nerve head surrounded by splinter hge.
- ❖ 1 - 2 months the swelling resolves & optic atrophy follows.

**Prognosis:** is very poor.

### Investigations:

- 1) **ESR:** Elevated usually above 60mm.
- 2) **CRP:** Positive.
- 3) **Temporal Artery biopsy:** Definite diagnosis.

### Treatment:

- ❖ It should be initiated rapidly as 65% of the untreated patients may become bilaterally blind in few weeks.
- ❖ The main aim of treatment is prevention of blindness in the fellow eye.

### Therapeutic regimen:

- 1) **Immediate treatment:** I.V. hydrocortisone 250 mg together with oral prednisone 80 mg/day.
  - ❖ Mega doses should be given when indicated under the supervision of internist.
- 2) **Subsequent treatment:**
  - ❖ After 3 days the dose is reduced to 60 mg for 3 days & 40 mg for 4 days.
  - ❖ The daily dose is reduced by 5 mg/week until 10 mg is reached.
- 3) **Maintenance dose:** 10 mg/day for 12 months, but the duration of treatment is variable governed by symptoms, Fundus examination & the level of ESR.

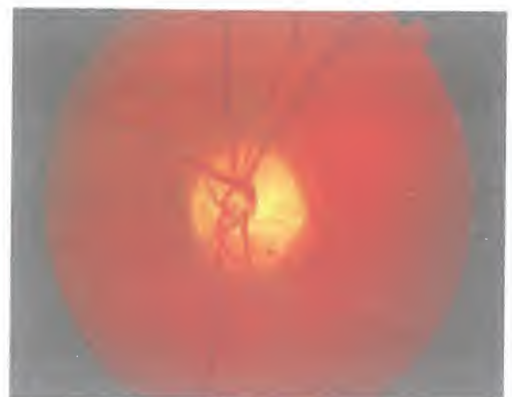


## 3) Optic Atrophy

**Definition:** It is a term applied to the condition of the disc when (- +) optic nerve fibers are degenerated due to interruption of the nerve fibers at any point between the ganglion cell layer & the lateral geniculate body.

### Etiology (types):

- 1) **Primary:** cause outside the eye:
  - ❖ **CNS disease:** Tabes dorsalis & Demyelinating diseases (commonest cause).
  - ❖ **Due to:**
    - a) **Trauma:** fracture base of skull.
    - b) **Tumor:** pituitary tumor.
    - c) **Ischemia:** severe blood loss.





2) **Secondary:** due to optic nerve diseases as: post-papilledemic & post-papillitic.

3) **Consecutive optic atrophy:** following a retinal disease as: CRAO, R. pigmentosa, degenerative myopia, amaurotic family idiocy.

4) **Glaucomatous optic atrophy:** due to glaucoma.

### Clinical picture:

**Symptoms:** ↓ of vision up to no PL.





### Signs:

1) **Pupil:** Unilateral afferent papillary defect (Paradoxical pupil = Marcus Gunn pupil)

Bilateral dilated fixed pupil.

2) **Fundus picture:**



	Primary	Secondary	Consecutive	Post -glaucoma
1) Disc				
a) Colour	Milky white	Grey white	Yellow waxy	Pale white
b) Edge	Well defined	Irregular	Irregular	Overhanging
c) Cup	Enlarged	Obliterated	Obliterated	Deeps,
d) Lamina	Seen	Not seen	Not seen	Seen
e) Vessles	Normal	Sheathed	Attenuated	Interrupted
2) Rest of retina	Normal	Normal	Show disease	Normal
				

### D.D.:

1) Other causes of optic atrophy.

2) Causes of gradual painless diminution of vision.

**N.B.:** The atrophic cup: its size is intermediate between physiological & glaucomatous with sloping edge.

**Treatment:** Mostly hopeless: Treatment of cause: Vaso-dilators.

# Multiple Sclerosis

- ❖ **Definition:** Immunologic disorder of the CNS with frequent visual complaints.
- ❖ **Incidence:** 6 to 8 per 100,000. (Women: Men → 2:1).
- ❖ **Age:** In young adults (25 - 40) years.
- ❖ **Pathogenesis:** Unknown but environmental and genetic factors may be involved.
- ❖ **Course:** MS is variable (often chronic with a relapsing course).
- ❖ **Manifestations:** Optic neuritis.
- ❖ **Investigations:** MRI.
- ❖ **Treatment of optic neuritis:**
  - 1) I.V. Methyl Prednisolone (250 mg/6h for three days) Followed by Oral Prednisone (11 dys).

# Migraine

## Definition:

- ❖ A clinical syndrome in which there are **attacks of headache frequently accompanied by visual and/or gastrointestinal symptoms.**
- ❖ The attack last for **several hours.**
- ❖ The severity of headache is variable and **may be associated with vomiting.**
- ❖ **In between attacks, the patient is completely free.**

## Clinical presentation:

### 1) Classical migraine:

- There are two phases, an aura lasting for 10 to 20 minutes, followed by headache.
- **The aura may involve:**
  - a) **Disturbed vision.** E.g.: Zig-zag lights, scotomas and hemianopic vision.
  - b) **Transient speech disturbance.**
  - c) **Tingling or weakness in the limbs on one side of the body.**

### 2) Headache only.

### 3) Visual or neurological symptoms only without headache.

### 4) Occasionally, the neurological features may start after the onset of the headache.

## Variants of the migraine syndrome:

### 1) **Hemiplegic migraine:** Accompanied by repeated attacks of unilateral paralysis.

### 2) **Ophthalmoplegic migraine:** Paralysis of the eye muscles with diplopia. The 3<sup>rd</sup> nerve is frequently affected.

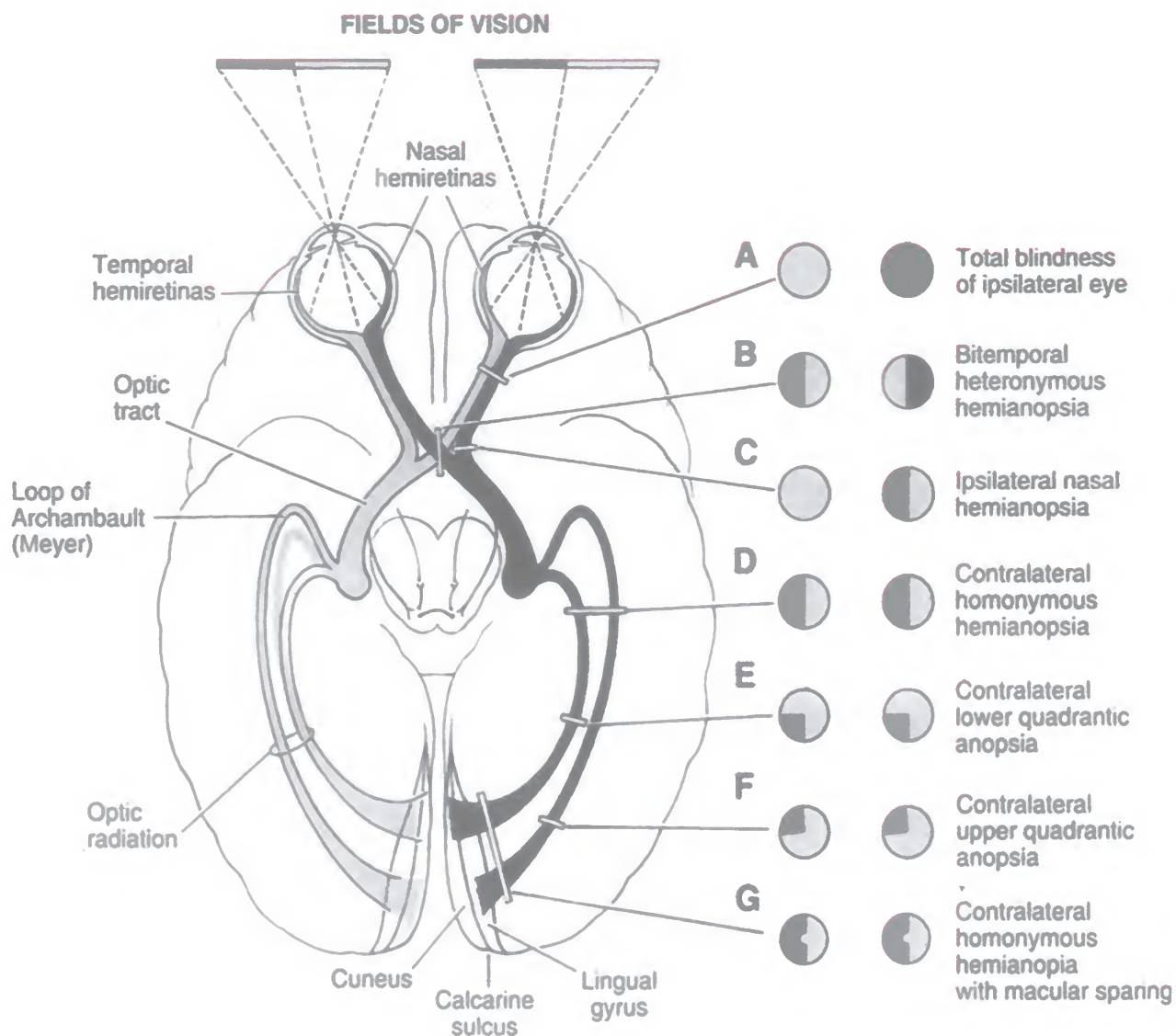
### 3) **Basilar migraine.**

### 4) **Migrainous neuralgia** (cluster headache).

### 5) **Status migrainosus**

# Neuro-

# Ophthalmology





# Anatomy of the Visual Pathway

- ❖ **The visual pathway** starts at the **photoreceptors (rods and cones)** in the retina. They synapse with **the bipolar cells**, which in turn synapse with **the ganglion cells**.
- ❖ **Axons of the ganglion cells** run in the nerve fiber layer of the retina and converge to form **the optic nerve (second cranial nerve)**.
- ❖ **Axons of the retinal ganglion cells** run in the optic nerve to **the optic chiasma** where:
  - The nasal fibers of each nerve decussate to reach the optic tract of the opposite side.
  - The temporal fibers pass uncrossed to the optic tract of the same side.
- ❖ **Each optic tract:** carries fibers from the temporal retina of the same side and nasal retina of the opposite side.

This arrangement will be respected to **the end of the visual pathway in the occipital cortex**.
- ❖ **Fibers of the optic tract** reach **the lateral geniculate body (LGB)** where they synapse.
- ❖ **Fresh axons from the LGB** spread out to form **the optic radiations**, which pass through a broad area of **the temporal and parietal lobes of the brain**.
- ❖ **Fibers of the optic radiation** end in **the visual cortex in the occipital lobe**. The visual cortex includes the primary visual area known as **the striate cortex (area 17)**, and **the visual association areas, pre -striate cortex (area 18)** and **para-striate cortex (area 19)**.
- ❖ **Each occipital cortex** receives stimuli from **the temporal retina of the same side** and **the nasal retina of the opposite side**.
- ❖ **Optic nerve** is not a true peripheral nerve, but rather a tract of the central nervous system.

Evidence for this including:

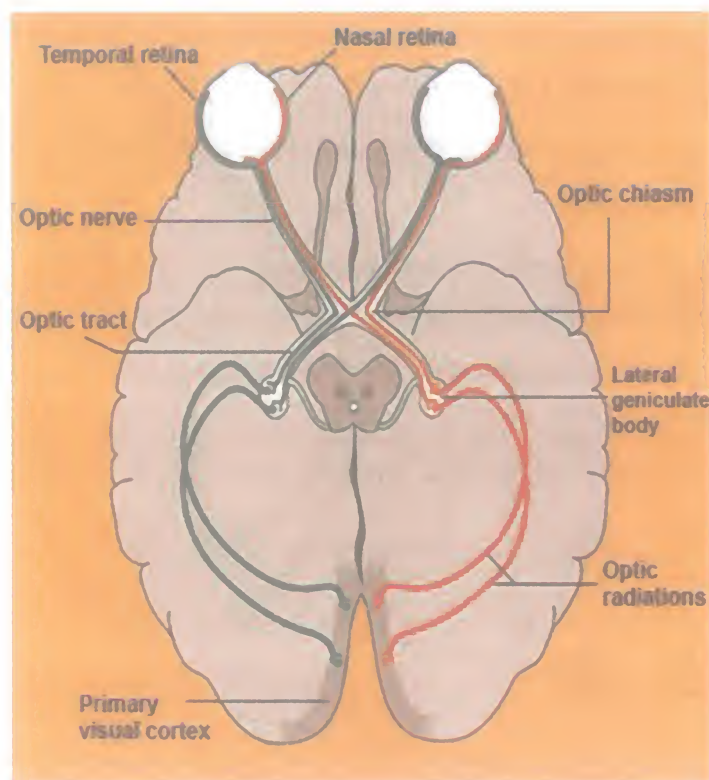
  - **Embryological:** optic nerve develops as a bud from the midbrain.
  - **Anatomical:** optic nerve is surrounded by the meninges and bathed in Cerebro-spinal fluid.
  - **Physiological:** fibers forming the optic nerve are axons of second –order neurons, which are found only in the CNS.
  - **Histological:** axons of the optic nerve have no neuro-lemmal sheaths, thus, they cannot regenerate.

# Neuro-Ophthalmic Manifestations of brain lesions

- 1) **Optic nerve lesions:** Monocular field defects on the same side, normal field on the other side.
- 2) **Optic chiasma:** Bitemporal hemianopia due to interruption of the nasal fibers of the optic nerve crossing at the chiasma. The commonest cause is **pituitary tumors**.
- 3) **Optic tract:** Contra lateral homonymous hemianopia that respect the vertical meridian.
- 4) **Optic radiations:**
  - a) **Temporal lobe lesions:** contra lateral homonymous superior quadrantic-anopia with hemiparesis and dysphasia.
  - b) **Parietal lobe lesions:** contra lateral homonymous inferior quadrantic-anopia with agnosia.
- 5) **Occipital cortex:** Contralateral homonymous hemianopia with macular sparing.

Macular sparing in occipital cortex lesions can be explained by:

  - a) **Dual blood supply of the macular area** (from the middle and the posterior cerebral arteries).
  - b) **Bilateral representation of the macular fibers.**
  - c) **Large area of macular representation in the occipital cortex.**



# The Pupil

## Pupillary Reaction:

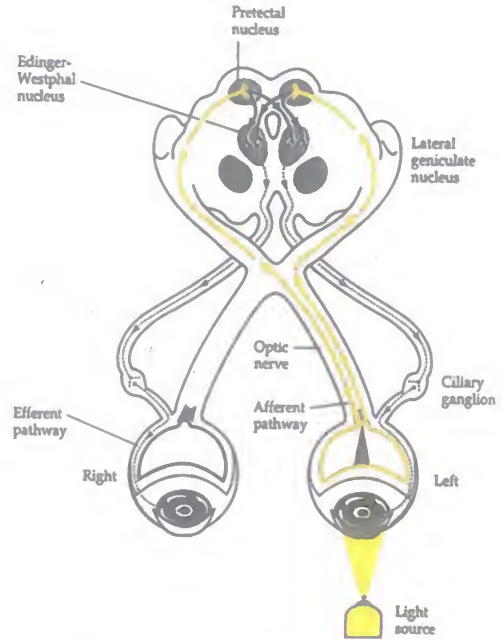
Activity of the parasympathetic (supplying the sphincter pupillae muscle) and sympathetic (supplying the dilator pupillae muscle) mediates changes in pupillary diameter.

## Light Reflex:

**Definition:** Constriction of the pupil when light is thrown on to the retina.

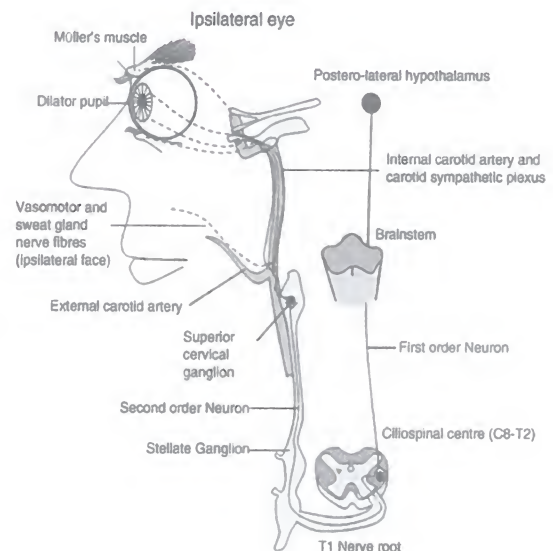
The light reflex has four neurons:

- 1) **The first:** starts at the photoreceptors in the retina, and ends in the **pre-tectal nucleus in the midbrain**, with nasal fibers crossing in the chiasma (first crossing).
- 2) **The second:** connects the **pre-tectal nucleus** to the **Edinger –Westphal nuclei** (of the third cranial nerve) **of both sides** (second crossing). This double crossing result in a direct and consensual light reaction of equal magnitude.
- 3) **The third:** runs in the oculo-motor nerve and connects the **Edinger- Westphal nuclei** to the **ciliary ganglion**.
- 4) **The fourth:** leaves the ciliary ganglion in the short ciliary nerves to **supply the sphincter pupillae muscle**.



## The sympathetic supply of the pupil:

- ❖ Starts at the **posterior hypothalamus** to the **cilio-spinal center of Budge at C8 - T1** in the spinal cord.
- ❖ **Pupillae-motor fibers** exit from the spinal cord and ascend with the sympathetic chain to synapse in the **superior cervical ganglion** to reach the **dilator pupillae muscle** with the **Naso-ciliary nerve** and **long posterior ciliary nerves**.



## The near reflex:

(Miosis, accommodation and convergence) occurs when looking at a near target.



# Abnormal pupillary reactions

## Relative Afferent papillary defect (RAPD): (Marcus-Gunn pupil):

- ❖ Lesions of the optic nerve result in **delayed transmission of impulses** and **weakness of the direct reflex and a normal consensual reflex**.
- ❖ The defect is d.t. a **unilateral optic nerve lesion or bilateral asymmetric optic nerve affection**
- ❖ It is best demonstrated by "the swinging flash light test".

## Argyll Robertson Pupils:

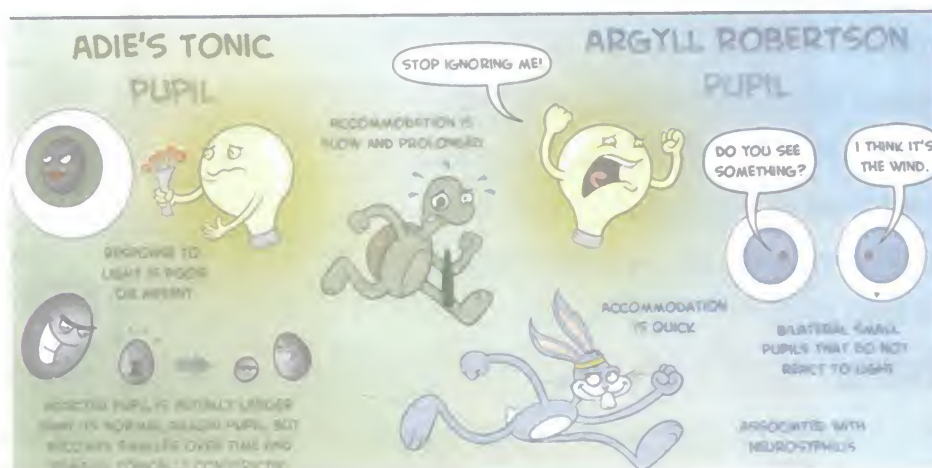
- ❖ The pupils are **small and respond poorly or not at all to light**, but have a prompt response to near (light – near dissociation).
- ❖ It occurs in **neuro-syphilis** due to a lesion in **the crossing midbrain fibers**.
- ❖ **Other causes include:** Diabetes, chronic alcoholism and trauma.

## Horner's syndrome (oculo-sympathetic palsy):

- ❖ **Homer's syndrome is caused by** a lesion in the sympathetic innervations to the eye.
- ❖ **Causes include:** Brain stem vascular or de-myelination diseases, bronchogenic carcinoma or nasopharyngeal tumors,
- ❖ **It is characterized by:**
  - 1) **Ptoxis** (due to weakness of the sympathetic innervated Muller's muscle).
  - 2) **Miosis** (due to the unopposed action of the sphincter pupillae).
  - 3) **Anhidrosis** (reduced sweating).
  - 4) **Enophthalmos**.

## Adie's Pupil:

- ❖ Damage to **the ciliary ganglion** or via **sympathetic fibers of the short post. ciliary nerves**.
- ❖ **The near reflex** is **tonic and segmental (light -near dissociation)**.
- ❖ **The condition is benign** and most commonly affects **women in their second to fourth decade** and may be associated with an **absent knee-jerk (Adie's-Holmes pupil)**.



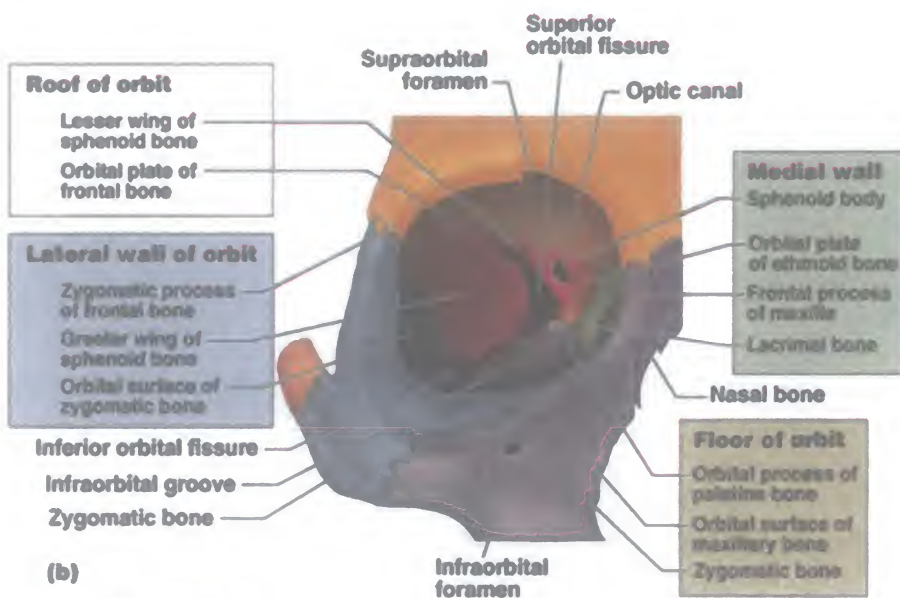
## Abnormalities of pupil size

- ❖ The normal pupil diameter averages between 3 – 4 mm.
- ❖ The size depends on the tone of the constrictor and the dilator muscles.
- ❖ There are many causes for miosis and mydriasis, some are **physiological**, and others are **pathological**.
- ❖ Some medications may cause miosis, others may cause mydriasis. (See Table).

### Causes of miosis and mydriasis:

	Miosis	Mydriasis
<b>Physiological (mobile)</b>	<ol style="list-style-type: none"> <li>1) Light reflex</li> <li>2) Accommodation reflex</li> <li>3) Corneo-pupillary reflex</li> <li>4) Sleep</li> <li>5) Senile</li> <li>6) Newly born</li> <li>7) 3<sup>rd</sup> stage of anesthesia</li> </ol>	<ol style="list-style-type: none"> <li>1) Withdrawal of light</li> <li>2) Cilia -spinal reflex</li> <li>3) Excitement</li> <li>4) Light-colored iris</li> <li>5) 2<sup>nd</sup> stage of anesthesia</li> </ol>
<b>Medications</b>	<ol style="list-style-type: none"> <li>1) Local miotics</li> <li>2) Opium</li> </ol>	<ol style="list-style-type: none"> <li>1) Local mydriatics</li> <li>2) Datura and atropine</li> </ol>
<b>Ocular causes</b>	<ol style="list-style-type: none"> <li>1) Acute iridocyclitis</li> <li>2) Trauma</li> <li>3) Paracentesis and hypotony</li> </ol>	<ol style="list-style-type: none"> <li>1) Acute glaucoma</li> <li>2) Trauma</li> <li>3) Blind eye</li> </ol>
<b>Neurological</b>	<ol style="list-style-type: none"> <li>1) Homer's syndrome</li> <li>2) Argyll Robertson pupil</li> <li>3) Pontine hemorrhage</li> <li>4) Irritative stage of cerebral compression</li> </ol>	<ol style="list-style-type: none"> <li>1) 3<sup>rd</sup> nerve paralysis</li> <li>2) Adie's pupil</li> <li>3) 4<sup>th</sup> stage of anesthesia</li> <li>4) Paralytic stage of cerebral compression</li> </ol>

# The Orbit





# Anatomy

The orbit is the socket of the eye.

It is pyramidal in shape with the base anterior and the apex posterior.

**The orbital volume is: 30 cm<sup>3</sup>:**

1) The globe occupies only about **one fourth** of this volume.

2) The lacrimal gland, optic nerve, extra -ocular muscles, vessels and nerves of the orbit occupy the remaining space.

3) The largest part is filled with fat.

## Orbital fissures and foramina:

1) **Optic foramen (canal):** It transmits: Optic nerve & Ophthalmic artery.

2) **Superior orb. fissure:** It transmits:

❖ 3<sup>rd</sup>, 4<sup>th</sup>, 6<sup>th</sup> cranial nerves.

❖ The 3 branches of ophthalmic nerve: Lacrimal, Frontal and Naso-ciliary.

❖ Sympathetic root of ciliary ganglion.

❖ Ophthalmic vein.

3) **Inferior orbital Fissure:** It transmits:

❖ Maxillary division of trigeminal.

❖ Infra -orbital artery.

# Diseases of the orbit

## Proptosis

**Definition:** Passive Protrusion of the eyeball = Globe pushed.

**N.B.: Exophthalmos = Active protrusion.**

## Etiology:

1) **Congenital:** (Dermoid cyst – Meningo-encephalocele).

2) **Traumatic:** e.g.:

❖ Retrobulbar hematoma.

❖ Surgical emphysema (rupture ethmoid bone with air escape).

❖ A/V shunts (carotid -cavernous fistula).

3) **Inflammatory:** e.g.:

❖ **Acute:** Orbital cellulites, cavernous sinus thrombosis & Panophthalmitis

**What are the causes of acute proptosis???**

❖ **Chronic:**

➤ **Specific:** T.B. granuloma.

➤ **Non-specific:** Orbital pseudo -tumor.

#### 4) Neoplastic:

a) **Systemic malignancy:** Lymphoma & Leukemia

b) **Metastases (secondaries):**

- **Systemic metastases:** Breast carcinoma (Female) – Bronchogenic & prostate carcinoma (Male) – Neuroblastoma of suprarenal (Children).
- **Local metastases from surrounding:** Tumors of the nose or sinuses

c) **Primary:** from orbital structures:

- **Bones:** Osteoma – Osteosarcoma
- **Muscles:** Rhabdomyoma – Rhabdomyosarcoma.
- **Lacrimal gland tumors:** Adenoma, Adenocarcinoma.
- **Optic nerve tumors:** Glioma, Meningioma.
- **Vessels:** Haemangioma, Hemangiosarcoma.

5) **Endocrinal:** Dysthyroid Ophthalmopathy (thyrotoxic exophthalmos & thyrotrophic).

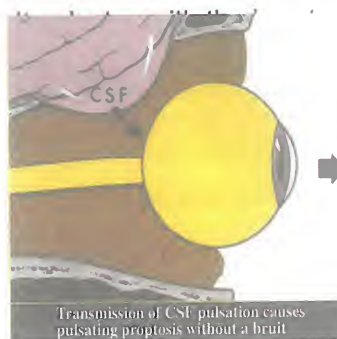
6) **Other causes:**

- ❖ **Vascular:** aneurisms, tumors, A/V shunts and varices.
- ❖ **Cyst:** Dermoid cyst, Hydatid cyst.
- ❖ **Paralysis:** of extra ocular muscle (3<sup>rd</sup> nerve palsy).
- ❖ **Bone disease:** Paget's disease



### Meningo-encephalocele

Herniation of the meninges and part of the brain through a defect in the orbital roof.



### Surgical emphysema

- ❖ Air passes from the nasal sinuses into the orbit and subcutaneous tissue of the lids through a fracture of the ethmoidal bone.
- ❖ Proptosis (increase on blowing nose) and crepitation (are felt on the eyelid).
- ❖ **Treatment:** Pressure bandage – Antibiotics – Don't blow the nose.

## Arterio-venous fistula:

**Cause:** It is due to rupture of internal carotid artery as it passes in the cavernous sinus following severe trauma → aneurysm.

### Clinical picture:

**Symptoms:** Severe pain due to stretch of the nerve branches & murmur.

### Signs:

- ❖ Pulsating exophthalmos which disappear on pressure on common carotid a.
- ❖ Dilated vessels of lid & conjunctiva.
- ❖ Fundus: distended veins

### Treatment:

- 1) Ligation of the common carotid artery in the neck.
- 2) Ligation of the artery before and after the fistula.

## Diagnosis:

### 1) History:

a) **Trauma:** To Orbital cellulitis.

b) **Pain (indicates inflammation).**

- Orbital cellulitis. →
- Cavernous sinus thrombosis.
- Panophthalmitis.

c) **Onset and course:**

- **Acute:** inflammation - trauma.
- **Intermittent:** varices.
- **Slowly progressive:** benign neoplasm.

### 2) Examination: before considering proptosis,

We should first exclude pseudo-proptosis which may be due to:

- ❖ Large globe
- ❖ Buphthalmos
- ❖ Shallow orbit as in cranio-facial anomalies
- ❖ Contralateral enophthalmos





**a) General examination:**

❖ **ENT examination.**

❖ **Medical examination:**

- Enlarged lymph nodes.
- Thyroid.
- Look for a primary.

**b) Local:**

**1) Inspection:**

❖ **Unilateral or bilateral:**

➤ **Bilateral:**

- ✓ Orbital reticulosis (lymphoma& Hodgkin's disease).
- ✓ Endocrinal.
- ✓ Late cases of cavernous sinus thrombosis.

➤ **Unilateral:** Other causes.

❖ **Direction:**

- Directly forward (e.g. optic Nerve tumors).
- Forward, down and in (e.g., lacrimal gland Tumors).
- Forward, down and out (e.g., frontal mucocele).
- Upward (e.g. cancer maxilla).

❖ **Pulsations:** e.g.: in:

- Ophthalmic Artery Aneurism.
- Vascular tumors (sarcoma).
- A/V shunt (carotid-cav. fistula).
- Meningo-encephalocele.

❖ **Signs of inflammation:**

- **Lid:** Edema.
- **Conjunctiva:** Chemosis, hyperemia.

**2) Palpation :**

- ❖ Consistency.
- ❖ Fixation of the lesion
- ❖ Tenderness.

**3) Auscultation:** In carotid cavernous fistula → machinery murmur (Bruit).

#### 4) Measurement of proptosis:

- ❖ The distance between the: Lateral orbital Margin & Apex of the cornea is measured.
- ❖ Normally: 15 – 17 mm.
- ❖ The measurement is done by: Simple ruler OR Hertels exophthalmometer.
- ❖ The best position to examine case of proptosis is from above and behind while the patient is seated

### 3) Investigations:

#### a) Laboratory:

- ❖ Complete blood picture e.g., for leukemia.
- ❖ Tuberculin test.
- ❖ T3, T4 and TSH levels.
- ❖ Casoni test.
- ❖ ESR

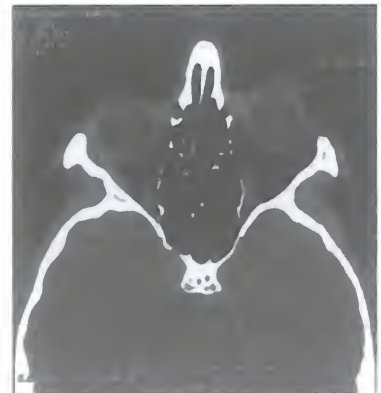
#### b) Radiological:

- ❖ Plain x-ray:
  - Wide optic Foramen (Glioma of optic nerve).
  - Calcification (Meningioma).
- ❖ Arteriography and venography (Carotid cavernous fistula).
- ❖ Ultrasonography.
- ❖ C-T scan.
- ❖ Magnetic resonance imaging (MRI).

#### c) Surgical: Biopsy.

### 4) Treatment:

- ❖ Orbitotomy and excisional biopsy.
- ❖ Treatment of the cause.
- ❖ Orbitotomy may be:
  - Anterior Orbitotomy: for lesions in the anterior  $\frac{1}{2}$  of orbit.
  - Lateral Orbitotomy: for deeper lesions.
  - Trans-frontal Orbitotomy: for orbital apex lesions (neurosurgery).
- ❖ Exposure (prophylactic).



# Important & Common Causes of Proptosis

## 1) Traumatic:

- a) Retro-bulbar hemorrhage.
- b) Surgical emphysema.
- c) Arterio-venous fistula (carotid -cavernous fistula).

## 2) Inflammatory:

- a) Orbital cellulitis.
- b) Cavernous sinus thrombosis.
- c) Panophthalmitis.

## 3) Endocrine: Dysthyroid ophthalmopathy.

# Orbital cellulitis

**Definition:** It is acute suppurative inflammation of the orbital cellular fibro-fatty tissue.

## **Etiology:**

### 1) Direct infection: by:

- ❖ Penetrating wounds.
- ❖ Following operation (squint. R.D.).

### 2) Spread of infection: from: Sinuses.

- ❖ Teeth.
- ❖ Globe (Endophthalmitis).

### 3) Blood borne: as in septicemia.

**Causative organisms:** Usually Staphylococci, streptococci, pneumococci and rarely Fungi.

## **Clinical picture:**

### Symptoms:

- ❖ **General:** Fever, malaise.
- ❖ **Local:** Pain.
- ❖ **Vision :**
  - Early: Good.
  - Late: Decrease due to optic Neuritis.



### Signs:

- 1) **Edema of lid and conjunctiva:** (Due to congestion in the ophthalmic vessels & vasodilatation of ophthalmic arteries)
- 2) **Proptosis** (suppuration in the orbit).
- 3) **Limitation of ocular motility** (myositis & optic neuritis → pain with movement).
- 4) **An abscess** may form & point through:
  - Lower fornix.
  - Skin near lower orbital Margin.

### **Complications:**

- 1) **Extension of infection into:**
  - ❖ Cranium (brain abscess, cav. thrombosis).
  - ❖ Optic Neuritis.
  - ❖ Globe → endophthalmitis.
- 2) **CRV thrombosis** (papilledema with engorged vs.)
- 3) **Enophthalmos** with restricted eye movement (due to healing by fibrosis) → (frozen orbit).
- 4) **Corneal exposure.**
- 5) **General spread** (septicemia & pyemia)

**D.D.:** See the table.

### **Treatment:**

- 1) **General:** Antibiotics.
- 2) **Local :**
  - ❖ Hot fomentation.
  - ❖ Incision: if abscess forms (drainage).
  - ❖ TTT of corneal exposure.



# The cavernous Sinus

It is one of the paired dural sinuses.

**Relations:** Lies lateral to sella turcica & medial to the temporal lobe.

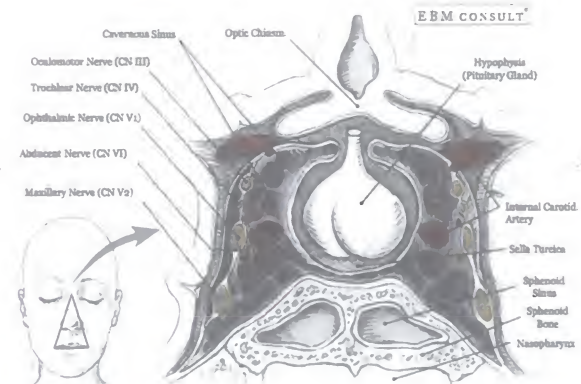
**Contents:**

## 1) The cavity contains:

- ❖ Internal carotid artery.
- ❖ Abducent nerve.

## 2) The lateral wall contains from above downward:

- ❖ 3<sup>rd</sup> nerve
- ❖ 4<sup>th</sup> nerve
- ❖ Ophthalmic nerve
- ❖ Maxillary nerve



**Communication:**

## 1) Anteriorly:

- ❖ Superior & inferior ophthalmic veins.
- ❖ Sometimes central retinal vein. Thus communicating with:
  - Eye & orbit.
  - Face: as ophthalmic veins communicate with angular veins.

## 2) Posteriorly:

- ❖ Superior & inferior petrosal sinuses thus communicating with:
  - Subcutaneous layer behind the ear:

**N.B.: Via the superior petrosal sinus & mastoid emissary veins.**

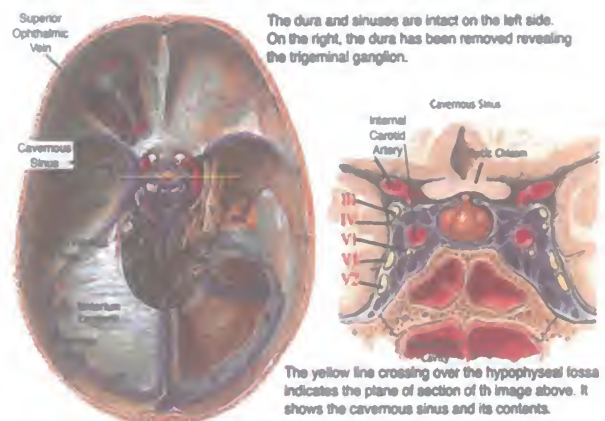
- ❖ Middle ear via inferior petrosal sinus & Jugular vein.

## 3) Superiorly: Middle cerebral vein.

## 4) Inferiorly: Emissary veins connecting it with pterygoid plexus which drain pharynx & mouth.

- ❖ Thus communicating with: The mouth, pharynx & nasal sinuses.

## 5) Medially: Intravenous sinuses to the other cavernous Sinus.



# Cavernous Sinus Thrombosis

**Definition:** Is thrombophlebitis of the cavernous Sinus.

**Etiology:** The infection may come from:

- a) **Face and orbit:** (ophthalmic vein).
- b) **Middle ear:** (infection petrosal sinus).
- c) **Mouth and pharynx:** (pterygoid plexus).
- d) **Blood borne:** (metastasis).

**Clinical picture:**

**Symptoms:**

- ❖ **General:** Fever, malaise (marked), headache anorexia (FAHM).
- ❖ **Local:** Pain.
- ❖ **Vision:** ➤ Early: Good.  
➤ Late: Decrease due to op. neuritis.

**Signs:**

- 1) **Lid and conjunctiva:** Edema.
- 2) **Proptosis.**
- 3) **Limitation of ocular motility** (paralysis of nerves in the sinus).
- 4) **Fundus:** engorged veins.
- 5) **Edema of mastoid region.**
- 6) **Pupil:** dilated with total ophthalmoplegia (paralysis of 3<sup>rd</sup>, 4<sup>th</sup>, 6<sup>th</sup> nerves).

**N.B.:** ❖ Extension to the other sinus → bilateral proptosis.

❖ The earliest sign in the other eye → convergent Squint (6<sup>th</sup> nerve palsy).

**Treatment:**

- ✚ The condition is very serious & could be fatal (from meningitis, brain abscess & pulmonary infection) if not well treated.

1) **Prophylaxis:** TTT of source of infection.

2) **Curative:**

- a) Massive antibiotics.
- b) Anticoagulant.
- c) Neurosurgical approach.
- d) Corneal protection from exposure keratitis.



**D.D.:**

	Endophthalmitis	Panophthalmitis	Orbital cellulitis	Cavernous S. thrombosis
<b>Definition</b>	It is suppurative Inflammation Primarily in uveal tract. (sclera is free)	It is severe suppurative inflammation of uveal tract and other tissue including the outer Coat & soft orbital tissues.	It is suppurative inflammation of orbital cellular tissue.	It is thrombophlebitis of the cavernous Sinus.
<b>Etiology</b>	As infective Iridocyclitis (see before)	As infective Iridocyclitis (see before)	See (the orbit)	See (the orbit)
<b>Cl. Picture:</b>				
<b>Symptoms:</b>				
<b>General:</b>	Headache, fever	More (+++)	(+++)	Severe (++++)
<b>Local:</b>				
<b>Pain:</b>	++	++	++	++
<b>Vision:</b>	decrease to no P.L.	no P.L.	good (early)	good (early)
<b>Signs:</b>				
<b>Lid</b>	Edema	Edema	Edema	Edema
<b>Conjunctiva</b>	Chemosis + ciliary injection	Chemosis + ciliary injection	Chemosis + injection	Chemosis + congestion
<b>Cornea</b>	Hazy + kps.	Hazy + ring abscess	Clear.	Clear.
<b>Proptosis</b>	Absent.	Present	Present	Present.
<b>Oc. Motility</b>	Normal	Limited	Limited	Limited
<b>Red reflex</b>	Yellow	Yellow	Normal.	Normal
<b>Treatment</b>	❖ Seeing eye → vitrectomy	Usually non seeing → evisceration	Orbital abscess → drain	Anticoagulant + drain + neurosurgical ttt
<b>Antibiotic +</b>	❖ non seeing → evisceration			

# Dysthyroid Ophthalmopathy (Thyroid eye disease)

## Definitions:

- ❖ Hyperthyroidism occurs in a number of diseases including: Grave's disease, toxic goiter, thyroiditis and excess intake of thyroid hormone.
- ❖ High levels of T4 give rise to weight loss, tachycardia, tremors, excessive sweating, nervousness, heat intolerance and palpitation.

## Grave's disease:

- ❖ A term used to describe the commonest variety of hyperthyroidism which has **an autoimmune basis.**
- ❖ It usually affects **females 20 - 45 years old and has characteristic eye features.**

## Ocular signs:

- ❖ Usually occur in the hyperthyroid state but they may appear also in a patient with normal thyroid function or even with subnormal function,
- ❖ The ophthalmic affection may precede or follow the hyperthyroidism.
- ❖ It is important that therapeutic control of hyperthyroidism may not improve, or may even worsen the Exophthalmos.



## Pathogenesis:

- Proliferation of orbital fat and connective tissue with retention of fluids, accumulation of mucopolysaccharides and cellular infiltration by lymphocytes and plasma cells.**
- Enlargement of the extra-ocular muscles** due to increased mucopolysaccharides and edema with subsequent degeneration of the muscle fibers, fibrosis, weakness and restricted ocular movements.

## Clinical features and Complications:

### 1) Eyelid signs:

- Dalrymple sign:** Lid retraction which gives a staring look.
- Von Graefe's sign (Lid lag):** The upper lid doesn't follow the eyeball when looking downward.
- Stellwag's sign:** Infrequent blinking.



## 2) Soft tissue signs:

- a) Injection, hyperemia and chemosis of the conjunctiva.
- b) Edema and fullness of the eyelid.

## 3) Proptosis:

- ❖ Dysthyroid eye disease is the commonest cause of both bilateral and unilateral proptosis.
- ❖ The condition may be very severe leading to exposure of the cornea with severe corneal ulceration and blindness.

## 4) Extra-ocular muscle affection: In the form of weakness and restricted movement in the form of:

- a) Defective **abduction**.
- b) Defective **adduction**.
- c) Defective **elevation**.
- d) Defective **depression**.

## 5) Optic neuropathy.

### **Management of thyroid eye disease:**

- 1) Local lubricants and dark glasses.
- 2) Treatment of corneal complications due to exposure.
- 3) Systemic steroids in early cases with painful proptosis.
- 4) Radiotherapy when steroids are contraindicated or ineffective.

## 5) Surgical:

- a) Orbital decompression in cases with painful proptosis.
- b) Extra-ocular muscle surgery when there is diplopia in the primary or reading position

### **Enophthalmos:** It is retraction of the globe into the orbit (opposite to proptosis)

- 1) Post-traumatic
- 2) Senile: absorption of orbital fat
- 3) Post-operative
- 4) Post-inflammatory
- 5) Homer's syndrome (ptosis – myosis – anhydrosis – enophthalmos)





# Operations of the orbit

1) **Orbitotomy:** To remove tumors or decompression in severe thyroid exophthalmos

## 2) **Enucleation:**

❖ **Principle:** The eye ball is excised, after taking the patient's consent

❖ **Followed by:** insertion of an artificial eye

❖ **Indications:**

- To stop pain: in absolute glaucoma.
- To save life: in intraocular malignancy (cut optic nerve as far as possible).
- To save other eye: in sympathetic ophthalmia.
- Cosmetic reasons: in total anterior staphyloma.

## 3) **Evisceration:**

❖ **Principle:** The cornea is excised and all the "contents" of the eyeball are evacuated, while the sclera is left. (after taking the patient's consent)

❖ **Indications:** Endophthalmitis and Panophthalmitis.

**N.B.: Here enucleation can't be done for fear of extension of infection along the sheath of optic nerve → brain.**

## 4) **Orbital exentration:**

❖ **Principle:** it is operation where all the contents of the orbit are removed inside the orbital periosteum. The lids may be also removed → all in one mass.

❖ **Indications:**

- Malignant orbital tumors.
- Malignant tumors of: Lid, Conjunctiva, Globe, (invading the orbit).

❖ **Contraindications:**

- Periosteum is invaded by the tumor.
- General metastasis.

# Eye Injuries

# Introduction

## Eye injuries Classified into:

- 1) Physical (mechanical injuries).
- 2) Chemical injuries.

**Physical (mechanical injuries):** May arise from many types of trauma, the most common:

- ❖ Blunt trauma.
- ❖ Penetrating traumas and foreign bodies.
- ❖ Radiation trauma.
- ❖ Ocular manifestations of head injuries.

## The normal protective mechanisms of the eye:

- 1) Eye lids & lashes.
- 2) Tear fluid (Mechanical & bactericidal).
- 3) Corneal sensation.
- 4) Bony orbit.
- 5) The cushioning effect of the retro-bulbar fat.
- 6) Bell's phenomenon.
- 7) Neck withdrawal reflex.
- 8) Const. of the pupil on exposure a strong light.

## Standard eye trauma terminology and classification:

**The eye wall:** Sclera and cornea

Though technically the eye wall has three coats posterior to the limbus, for clinical and practical purposes violation of only the most external structure is taken into consideration.

1) **Closed globe injury:** No full-thickness wound of eye wall

a) **Contusion:** There is no full thickness

The injury is either due to **direct energy delivery by the object** (e.g. choroidal rupture) or **the changes in the shape of the globe** (e.g. angle recession)

b) **Lamellar laceration:** Partial-thickness wound of the eye wall.

2) **Open angle injury:** Full-thickness wound of eye wall.

a) **Rupture:** Full-thickness wound of eye wall, caused by a blunt object.

b) **Laceration:** Full-thickness wound of eye wall, caused by a sharp object.

The wound occurs at the impact site by an outside-in mechanism:

❖ **Penetration injury: Entrance wound.**

If more than one wound is present, each must have been caused by a different agent.

❖ **Intraocular foreign body (IOFB): Retained foreign objects.**

Technically a penetrating injury, but grouped separately due to different cl. Implication.

❖ **Perforating injury: Entrance and exit wound.** Both wound caused by the same agent.



# Blunt trauma

**Cause:** Trauma by a small blunt object e.g.: Fist or Tennis ball.

## **Mechanism of damage:**

- 1) **Coup & countercoup:** coup refers to local damage at the site of impact (e.g. corneal abrasion) while counter coup refers to distant damage caused by shock rebound waves that traverses the eye to the posterior pole (e.g. commotio retinae).
- 2) **Antero-posterior:** compression & horizontal expansion this lead to damage (e.g. rupture globe & irido-dialysis).



# Effects of blunt trauma

## **Orbit:**

### 1) **Traumatic proptosis:**

#### ❖ **Cause:**

- Retro-bulbar hematoma.
- Peri-bulbar air (surgical emphysema due to fracture ethmoid sinus).



### 2) **Traumatic enophthalmos:**

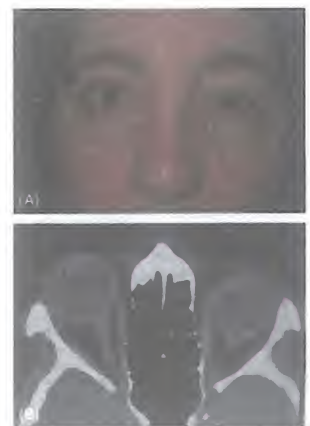
- ❖ **Cause:** Fracture of the orbital floor (blow out fracture) with escape of the orbital fat & extra ocular muscle into → the maxillary sinus.

#### ❖ **Sign and symptoms of a blow-out fracture:**

- Pre-orbital emphysema (air in the lids) is often present. It is caused by communication between the orbit and Peri-orbital sinuses.
- Diplopia and defective eye movement, usually elevation, due to entrapment of an extra-ocular muscle in the fractured site.

#### ❖ **Management of a blow-out fracture:**

- Computed tomography (CT scan) of the orbit
- A blow-out fracture rarely requires immediate surgery.
- Oral antibiotics (to protect against sinus bacteria and development of orbital cellulitis)
- Surgery will aim at correcting persistent problems as diplopia and disfiguring enophthalmos.



### 3) **Orbital cellulitis.**



## Lids:

### 1) Ecchymosis: Subcutaneous hematoma

(Black eye → cold foment in 1<sup>st</sup> 24 hours  
& hot foment thereafter)

### 2) Surgical emphysema.

### 3) Ptosis:

#### ❖ Causes:

- **Mechanical**: due to the weight of the lid (hematoma).
- **Paralytic**: due to injury to the levator - 3<sup>rd</sup> nerve.

### 4) Wounds: (treatment Suture in layers)

- ❖ **Vertical**: Heals by excessive scarring → cicatricial ectropion.
- ❖ **Horizontal**: Heals by less scarring.

## Conjunctiva:

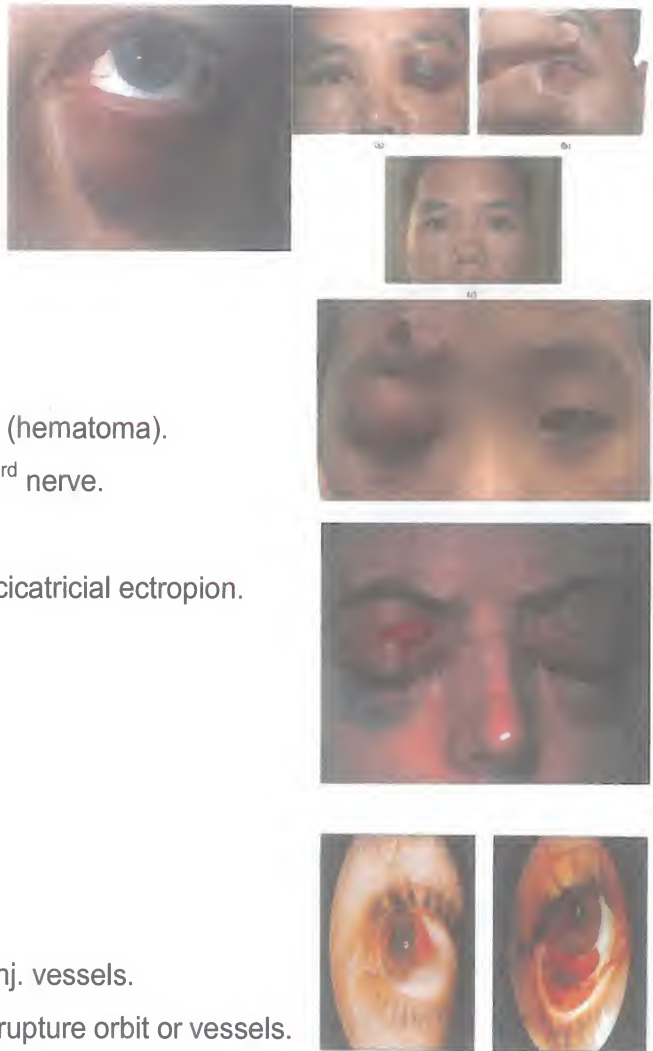
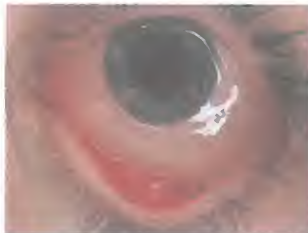
### 1) Wounds:

- ❖ If small (< 1 cm) → Leave it.
- ❖ If large → Suture it.

### 2) Sub-conjunctival Hge: may be due to:

- ❖ Direct trauma to the eye with rupture conj. vessels.
- ❖ Trauma to the head (fracture base) with rupture orbit or vessels.

### 3) Chemosis.



	Ocular Trauma	Head Trauma
1) Onset	Immediate	Delayed
2) Trauma	To the eye with no proptosis	To the head with proptosis
3) Consciousness	state Not affected	Lost
4) Site	On the temporal side	In the fornices
5) Color	Bright red	Dark red
6) Shape	Triangular with the base toward cornea	Triangular cornea (apex → cornea)
7) Color	Bright red	Dark red
8) Posterior limit.	Seen (defined)	Not

## **Cornea:**

### **1) Corneal foreign body:**

- ❖ Corneal foreign bodies are removed from the surface of the cornea by using a **foreign body spud after instilling topical anesthesia**.
- ❖ **Topical antibiotic drops and ointment** are then instilled with **patching of the eye**

### **2) Abrasion:**

- ❖ **Cause:** Damage to the corneal epithelium.

### **3) Wounds:** (= Rupture Globe with or without iris prolapse).

- ❖ **Treatment:** Suture the wound & reposit or excise the prolapsed tissues.

### **4) Blood staining of the cornea:**

- ❖ **Cause:** Hyphema + Rise in IOP.
- ❖ **Clinical picture:**
  - **The color of the cornea is first:** reddish brown then → greenish yellow → Grey.
  - The condition usually clears from the periphery → center by phagocytic action (Over 2 years or more).
- ❖ **Treatment:**
  - **Prevention:** Control of IOP in cases of hyphema (anti-glaucoma drugs or paracentesis).
  - **Treatment of hyphema.**
  - **Curative:** P.K. if Blood staining becomes Permanent.

### **5) Corneal Edema:**

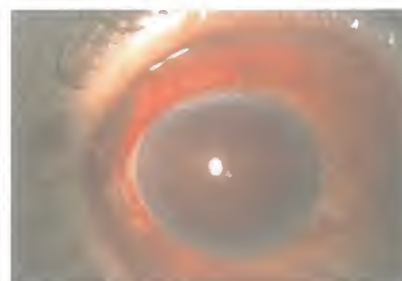
- ❖ **Cause:** due to endothelial & Descemet's membrane damage.
- ❖ **Treatment:** Mild → hygroscopic drops (glycerin).

### **6) Recurrent corneal erosion: (see cornea)**

**Sclera:** Laceration (Rupture Globe):

#### **1) Rupture Globe:**

- ❖ **Site:**
  - **Cornea:** Less common as the cornea is stronger than the sclera.
  - **Limbal:** Weak due to the presence of canal of Schlemm.
  - **Sclera:** Commonly up & In (about 3 mm from the limbus) Why?
  - **Trauma:** Is common from down & out (less protected).
  - **Eye:** Is pushed against the trochlea.



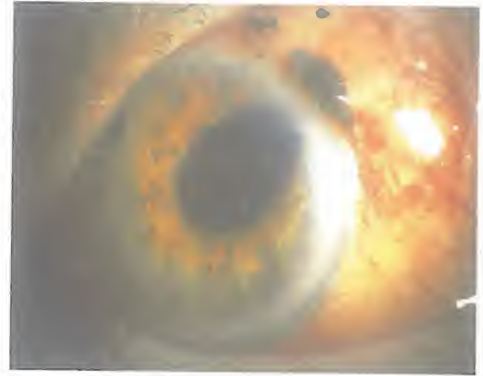


## 2) Signs & symptoms:

- ❖ Diminution of vision.
- ❖ Chemosis of the conjunctiva or sub-conjunctival hemorrhage.
- ❖ Shallow A.C.
- ❖ Hypotony.
- ❖ Abnormal site, size and shape of the pupil.
- ❖ Prolapse of parts of the uvea.

## 3) Complications:

- ❖ Infection (endophthalmitis).
- ❖ Intra-ocular Hge.
- ❖ Prolapse of intra-ocular contents → Iris, vitreous.
- ❖ Sympathetic ophthalmitis.
- ❖ Retina as RD.
- ❖ Lens: extrusion, rupture or dislocation.



## 4) Treatment:

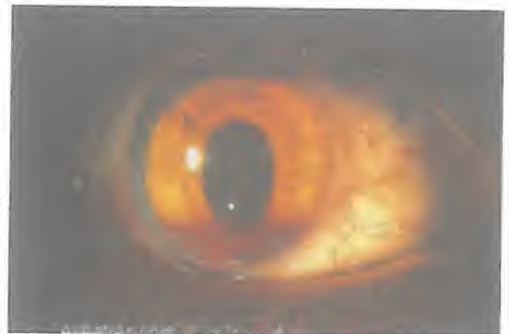
- ❖ Immediate patching & direct pressure should be avoided.
- ❖ Medical treatment: Systemic Antibiotic, Anti-tetanic-toxoid, Anti -emetic.
- ❖ X-ray is done to exclude the presence IOFB.
- ❖ Surgery:
  - Hopeless (no PL vision) → Enucleation
  - Hopeful → Reposite or excise the prolapsed tissues with proper suturing of the wound.

## **Anterior chamber**

### 1) Anterior Dislocation.

### 2) Hyphema:

- ❖ **Source:** From lacerated iris & CB.
- ❖ **Complications:**
  - 2ry glaucoma.
  - Blood staining of the cornea.
- ❖ **Treatment:**
  - Rest in bed (in a semi-sitting position).
  - Bandage of both eyes
  - Daily monitoring of the IOP



➤ **Medical treatment:**

a) **Local:** Steroid &  $\beta$ -blocker (control IOP).

b) **General:** Antifibrinolytic as Aminocaproic acid (50 – 100 mg/kg) every 4 hours to avoid rebleeding + Vit. C & Chemotrypsin.

c) **(NSAID are avoided).**

➤ **Surgery:** for the 2ry glaucoma:

a) **Paracentesis.**

b) **Evacuation of Hyphema:** washing (I/A).

**N.B.:**

❖ **No Mydriatics as they:**

➤ Lead to decrease absorptive surface of the iris.

➤ Lead to angle closure glaucoma.

❖ **No Myotics as they:** Lead to pupil block glaucoma & synechia.

❖ **Both lead to** iris movement with loosening of the clot → re-bleeding.

**The pupil:**

1) **Traumatic Miosis (mild trauma):** Mild trauma due to Iridocyclitis → spasm of the sphincter ms.

2) **Traumatic Mydriasis (severe trauma):**

❖ It is preceded by Miosis & may be Transient or permanent.

❖ It is due to damage of motor nerves (3<sup>rd</sup> nerve).

3) **Adie's pupil:** (see Neuro)

**The iris:**

1) **Laceration (Sphincter tear):**

❖ At pupil → **v-shaped** (Mydriatics are contra-indicated → ↑ tear).

❖ Pupillary laceration of iris may be accompanied with:

➤ **Anti -flexion:** Torn part is rolled anterior & pigment epithelium faces the anterior chamber.

➤ **Retro-flexion:** Torn part is rolled backward between lens equators ciliary body.

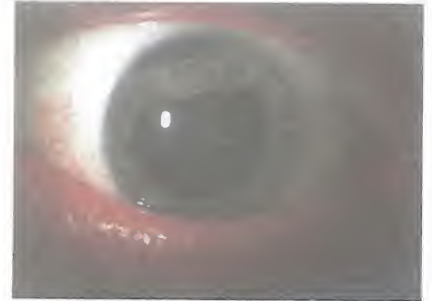
2) **Traumatic Iridocyclitis**

3) **Irido-donesis (Tremulous Iris):** Occurs with → Subluxation – Dislocation of lens.



#### 4) Irido-dialysis or Cyclo-dialysis:

- ❖ **Definition:** It is separation of iris root from C.B. This results from pressure In A.C. pushing iris backward. The iris periphery being not supported by lens may separate.
- ❖ **Symptoms:** Unocular diplopia except if:
  - Up (covered by the lid) **OR** Very small.
- ❖ **Signs:**
  - (Pupil → **D-shaped**) (Red reflex → double).
- ❖ **D.D.:** From malignant melanoma of the iris (RR → normal).
- ❖ **Treatment:**
  - **If no diplopia:** Atropine + cortisone.
  - **If diplopia exists:**
    - ✓ Cover the defect (colored C.L.).
    - ✓ Close the defect → suture the dialyzed iris to limbus.
    - ✓ Iridectomy.



- 5) **Traumatic Aniridia:** Here, complete avulsion of the iris at its root & the iris falls in the bottom of A.C. (as a black ball).

#### **Ciliary Body:**

- 1) **Hypotony:** Due to CB shock or Cyclo -dialysis.
- 2) **Glaucoma:** Due to CB laceration:
  - a) Hyphema.
  - b) Healing by fibrosis closing the angle (angle recession glaucoma).
- 3) **Spasm of accommodation:** With temporary myopia
- 4) **Paralysis of accommodation:** With blurring of near vision.
- 5) **Intra-Ocular Hge:** Hyphema, vitreous Hge.

#### **Choroids:**

- 1) Choroidal Effusion or Hge.
- 2) **Choroidal Rupture:**
  - ❖ **Site:** Temporal to the disc.
  - ❖ **Shape:** Crescentic with its concavity toward the disc.
  - ❖ **Color:** White (showing the sclera).
  - ❖ **Crossed by:** The retinal vessels (Retina is intact).
  - ❖ **Fate:** Good if away from the fovea.
  - ❖ **Treatment:** Rest & Vit. C.
- 3) Traumatic choroiditis.
- 4) Choroidal detachment from Hypotony.





## **Lens:**

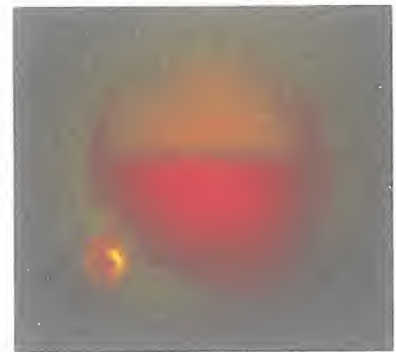
- 1) Subluxation.
- 2) Dislocation.
- 3) Traumatic Cataract (with Vossius ring).

## **Vitreous:**

- 1) Vitreous Hge.
- 2) **Vitreous Opacification: Musca volitans.** Due to:
  - a) Hemorrhage.
  - b) Coagulated protein.
- 3) **Vitreous Loss:** through a ruptured globe R.D.
- 4) **Avulsion of the vitreous base:** causing retinal dis-insertion.

## **Retina:**

- 1) **Retinal Tears** (Dialysis or giant tear) → RD.
- 2) **Retinal hge:** Intra-retinal, subhyaloid. →
- 3) **Retinal edema** (Comotio retinae = Berlin's edema).
- 4) **Retinal Detachment may be:**
  - a) **Rhegmatogenous:** due to retinal tear.
  - b) **Exudative:** due to severe Hypotony.
  - c) **Tractional:** due vitreous Loss & incarceration in scleral wound.



## **Comotio Retinae (Berlin's edema)**

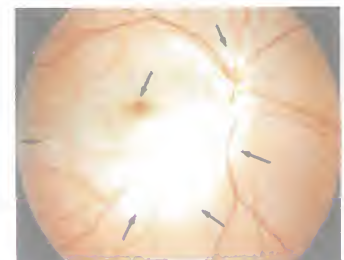
**Definition:** It is retinal edema following blunt trauma.

**Cause:** A countercoup to the posterior pole of the eye → compression of the posterior pole vessels  
→ retinal Edema (most marked in the central part).

**Symptoms:** Rapid drop of vision (HM).

### **Signs:**

- ❖ Pupil sluggish.
- ❖ **Fundus exam:** shows cherry red spot (+ Ret. hge).
- ❖ **White color** (due to edema collected mainly in the ganglion cell layer).
- ❖ **The fovea (no ganglion cells)** → Still show the color of the choroid.



### **Fate:**

- ❖ Resolution.
- ❖ Macular degeneration or hole.

**D.D.:** From other causes of: **Cherry red spot & Rapid diminution of vision.**

**Treatment:** Rest (if severe systemic steroids).

### **Optic Nerve:**

- 1) Avulsion of the optic nerve with twisted injuries.
- 2) Optic nerve Hge.
- 3) Edema of optic nerve with Hypotony.
- 4) Optic atrophy (1ry).

### **IOP:**

- 1) Traumatic glaucoma (cause).
- 2) Traumatic Hypotony (C.B. shutdown). ❖ Extra-ocular muscles: Squint.

### **Medico-legal aspects of blunt trauma**

- 1) Lacerations of the pupil.
- 2) Traumatic mydriasis.
- 3) Rosette cataract.
- 4) Vossius ring
- 5) Retinal dialysis.
- 6) Post-traumatic macular hole, macular pigmentation and choroidal rupture.

## **Perforating Trauma**

❖ **Causes:** Trauma by sharp instruments (knife, scissors).

❖ **Effects:**

- 1) **Mechanical effects:** Cut wounds in lid, cornea, sclera, lens capsule + uveal prolapse or vitreous loss.
- 2) **Infection:** Usually ends in Endo, Or even Panophthalmitis.  
➤ Onset: 24 – 48h (Bacterial) or weeks (Fungal)
- 3) **Sympathetic ophthalmitis.**

# Sympathetic ophthalmitis

**Definition:** It is **bilateral inflammation of the uveal tract** following perforating trauma to one eye in which part of the uveal tract is involved leading to marked diminution of vision.

- The traumatized eye is called (**the exciting eye**).
- The other eye called the (**sympathizing eye**).

**Etiology:** Predisposing Factors: The incidence is ↑ with:

- ❖ Injury to the CB (dangerous zone).
- ❖ Retained IOFB.
- ❖ Incarcerated uveal tissue in the wound.

## **Theories:**

- ❖ **Allergic theory:** The uveal tissue is normally isolated from the immune system of the body. An ocular injury will liberate the uveal pigment (Ag) to the circulation, The Ab formed against the uveal pigment will attack both eyes.
  - This theory is the most accepted one since:
    - 1) The latent period of the disease is about 4 – 8 weeks, which is the average time needed by the immune system to synthesize antibodies.
    - 2) Patients with sympathetic ophthalmitis have **cutaneous hypersensitivity to the uveal pigments**
    - 3) The disease is **extremely rare to occur if ocular suppuration takes place** due to destruction of the uveal pigments by pus.
- ❖ **Infective theory:** In which the organism (may be virus??) reach the exciting eye with the trauma & reach the other eye via Optic nerve → chiasma → other optic nerve.

## **Clinical picture:**

**Onset:** 4 – 8 weeks after the trauma (may be up to 30 years).

**Prodromal picture:** Sympathetic irritation better seen in the sympathizing eye.

## **Symptoms:**

- ❖ Pain, Lacrimation, Photophobia & Defective vision.
- ❖ Loss of accommodation (Indistinct near object) symptoms are bilateral but **start in the exciting eye** followed (days -weeks) by the sympathizing eye.

## **Signs:**

- ❖ Signs of **trauma in the exciting eye**.
- ❖ Signs of **bilateral iridocyclitis (KPs)** with variable degree of severity.



**Full picture:** The condition progress into:

- ❖ Sever bilateral pan-uveitis.
- ❖ 2<sup>ry</sup> Glaucoma, Complicated cataract, RD.
- ❖ Finally → Atrophia bulbi.

**Treatment:**

**1) Prophylactic treatment:**

- ❖ If the injured eye is hopeless: Do enucleation.
- ❖ If the injured eye is hopeful: The following is done:
  - Excise the prolapsed tissues.
  - Remove any IOFB.
  - Proper suturing of the wound + Follow up.

**2) Curative treatment:**

- ❖ If the Prodromal symptoms & signs appear in the normal eye give:  
**Topical Atropine & Cortisone.**
- ❖ Enucleate the traumatized eye (help to ameliorate the condition).

**N.B.:** Sympathetic ophthalmitis never occur if there is suppurative choroiditis (Endo or Panophthalmitis) as suppuration will destroy the uveal pigment (Ag).

## Injuries by Foreign Body

**Cause:** The foreign body may be: Iron, copper, lead, piece of glass, etc.

## Extra-ocular F.B.

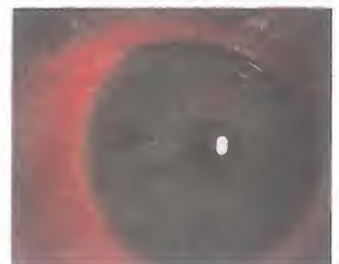
**Commonest sites:** Cornea - Fornix - Sulcus subtarsalis.

**Complications:**

- a) Corneal ulcers.
- b) Corneal opacities (If Bowman Membrane is damaged).

**Treatment:** Removal (under surface anesthesia):

- a) Use a glass rod over its tip is wrapped a piece of cotton and sweep the corneal surface.
- b) If embedded use a needle (or F.B. spud).



# Intra-Ocular F.B.

## Effects:

### 1) Mechanical effects: This depends on:

- ❖ **Route of entry** (cornea or sclera).
- ❖ **Size** (large F.B. → more damage).
- ❖ **Shape** (ragged F.B. → more damage).
- ❖ **Velocity**.

### 2) Infection: Relatively less common than in perforating traumas as **many IOFB are sterile by the heat generated during their emission.**

- ❖ **Sterile Inflammation** is common especially with **pure Copper**.

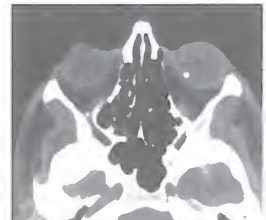
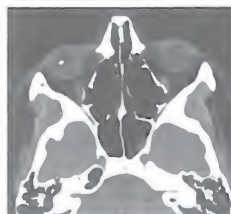
### 3) Sympathetic Ophthalmitis.

### 4) Chemical effects:

- ❖ If the F.B is **chemically inert (Glass)** → it will be **surround by fibrosis**.
- ❖ If **chemically active**:
  - Iron: Siderosis.
  - Copper: Chalcosis.

## Diagnosis & localization:

- ❖ **History.**
- ❖ **Slit lamp:** to detect: Route of entry ---- F.B in the AC or lens.
- ❖ **Gonioscopy:** to detect F.B hidden in the angle.
- ❖ **Ophthalmoscopy:** to detect F.B in the posterior segment (Retina, vitreous).
- ❖ If the media is **opaque** (due to: hyphema, cataract, vitreous Hge) use:
  - Plain X-ray.
  - X-ray using metallic limbal ring.
  - Ultra-sonography.
  - C.T. scan.



## Treatment: Removal of the F.B. (Via the nearest way to minimize the damage)

- ❖ **F.B in AC or on the iris:** Removed through a limbal incision using (Magnet or Forceps).
- ❖ **F.B entangled in the iris:** Do iridectomy.
- ❖ **F.B in the lens:** Do lens extraction
- ❖ **F.B in the posterior segment:** Removed through pars plane incision using:
  - Giant Magnet.
  - Vitrectomy + Forceps extraction.
- ❖ **Treatment of any associated damage.**

## Siderosis bulbi

### Definition:

- ❖ It is the toxic effect of iron on the eye.
- ❖ **Etiology:** Iron IOFB – Intra-ocular Hge.

### Mechanism & Clinical Picture:

- ❖ **Iron is oxidized** (rust → ferrous & ferric oxide) & the rust separates from the F.B & dissolve in the tissue fluids & circulate inside the eye leading to:
  - a) Stain the tissues with a **rusty (brown-red) color**.
  - b) Then, enters the cells (especially the epith. cells) producing toxic effect on the cellular protein & inactivates intracellular oxidative enzymes leading to **symptoms of diminution of vision & night blindness**.
  - c) Deposited in the angle scarring of the TM → **2<sup>ry</sup> glaucoma**

### Signs:

- ❖ **Cornea:** krukenberg spindle (iron in endothelium).
- ❖ **Iris & C.B:** Atrophic changes (Mydriasis) & Heterochromia.
- ❖ **Lens:** Cataract (not true cataract).
- ❖ **A.C. angle:** 2<sup>ry</sup> OAG
- ❖ **Retina (rods):** pseudo-retinitis Pigmentosa (night blindness).
- ❖ **Optic N:** Consecutive optic atrophy (Blindness).

### Treatment:

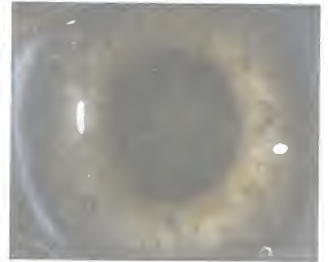
Early removal of the F.B. & treatment of complications: Cataract – Glaucoma.

## Chalcosis bulbi

**Definition:** It is the toxic effect of copper on the eye.

### Mechanism:

- ❖ **Pure copper:** produce severe inflammation that simulates endophthalmitis.
- ❖ **Copper is oxidized** into CuO which separates from the F.B → dissolve in the tissue fluids & circulate inside the eye leading to → staining of ocular tissues (especially collagen & basement membranes) with a yellow-green color.
  - **Lens:** Sun-flower cataract.
  - **Cornea:** Kayser-Fleischer ring (golden ring at the corneal periphery).





# Radiation Injuries - physical

## 1) Infra-red rays (longer wavelenght):

- ❖ **Effect:** Glass blower cataract & exfoliation of the anterior capsule (Iron melting cataract).
- ❖ **Prevented by:** Protective goggles.

## 2) X-ray: **Effect:** Madarosis, cataract, retinopathy, optic neuropathy.

## 3) Ultra-violet rays (shorter wavelenght):

- ❖ **Effect:** Photophthalmia.
  - It is Keratoconjunctivitis 2<sup>ty</sup> to exposure to short wave lengths.
  - As in: Sking (snow blindness) ---- Welding arcs.
- ❖ **Clinical picture:** Pain, lacrimation, severe photophobia (punctate corneal erosion)  
(Occur after a latent period of 6 – 8 h & last 12 – 24 h till the epithelium heals).
- ❖ **Treatment:**
  - **Prevention:** (protective goggles).
  - **Curative:** Bandage + Antibiotic drops.

## 4) Excessive light:

- ❖ **Effect:**
  - **Transient blindness** → due to saturation of visual receptors.
  - **Permanent damage:** Looking directly to the sun (solar ray in sun eclipse).  
This is due to absorption of light by RPE → heat → macular burn, scar or hole.

# Chemical injuries

## **Cause:** May be due to:

- |   |                               |
|---|-------------------------------|
| 1) Strong acids (e.g. H <sub>2</sub> SO <sub>4</sub> ). | 2) Strong alkalis (e.g. KOH). |
| 3) Lime.  | 4) Iodine.                    |
| 5) Aniline dye (e.g. from aniline pencils).             |                               |

## **Clinical picture:** Pain, tearing, photophobia and diminution of vision.

### 1) Mild to modearte exposure:

- Eyelid edema.
- Chemosis.
- Conjunctival injection.
- Corneal abrasions.
- Anterior uveitis.



## 2) Severe exposure:

- a) Conjunctival and episcleral whitening (coagulative necrosis).
- b) Corneal edema and opacification with corneo scleral melting.
- c) Severe iritis.
- d) Secondary glaucoma.
- e) Posterior segment destruction.

## **Complications:**

3) Ulceration: of skin, conjunctiva & cornea.

4) 2ry infection.

5) Scarring:

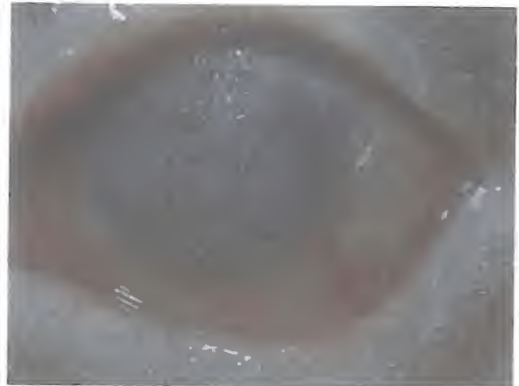
- ❖ **Lid:** Cicatricial entropion or ectropion.
- ❖ **Conjunctiva:** Symblepharon, xerosis.
- ❖ **Cornea:** Opacities, xerosis.

6) 2<sup>ry</sup> Glaucoma due to:

- ❖ **Iritis** (in alkali burn).
- ❖ **Conj fibrosis** → including the aqueous veins.

7) Atrophia bulbi (loss of vision): This depends on:

- ❖ **Concentration.**
- ❖ **Duration:** interval between chemical trauma & 1<sup>st</sup> aid.
- ❖ **Penetration:** Acids are less serious than alkalis because they coagulate & precipitate protein. Which act as a barrier that prevents further penetration (superficial burns).



## **Treatment:**

1) First Aid treatment:

✚ If the nature of the chemical substance is known → Wash the eye with the proper antidote as:

- ❖ **Strong acids** → Wash with  $\text{NaHCO}_3$  3%.
- ❖ **Strong alkalis** → Wash with Boric acid lotion 4%.
- ❖ **Iodine** → Wash with starch solution or milk.
- ❖ **Lime:**
  - a) Pick the particles with forceps.
  - b) Wash with:
    - EDTA (0.1 %).
    - Never with water.
    - Concentrated sucrose solution → neutral lime saccharate.

❖ Aniline → wash with:

➤ Alcohol 10%.

➤ Fluorescein then 10% glycerin.

✚ If the nature of the chemical substance is unknown: Wash the eye with either:

❖ Tap water → to dilute the chemical substance.

❖ Milk: Dilution – Buffers acids & alkalis.

Form a superficial film which protects the underlying tissues.

2) Conjunctival PH is detected:

Using litmus paper after irrigation is stopped (we should reach the neutral PH)

3) Drugs: (Topical & General)

❖ Atropine: for corneal ulceration.

❖ Antibiotic: against 2ry infection.

❖ Vitamins (A & C).

4) Prevention of symblepharon (Glass rod).

5) Therapeutic Keratoplasty (for impending perforation).

6) Treatment of complications.

## Ocular emergencies

1) Chemical injuries, perforating injuries (rupture globe), IOFB.

2) Closed angle glaucoma

3) Anterior lens dislocation.

4) CRAO.

5) Acute redness of the eye (ciliary injection).

6) Widely spreading inflammatory conditions (Endophthalmitis, Panophthalmitis, Orbital cellulites, Cavernous sinus thrombosis)

7) RD. with macula threatened.



# Head injuries

## 1) Visual pathway lesion at various levels:

(Optic nerve – Optic chiasma – Optic tract – L.G.B. – Optic radiation – Occipital cortex).

## 2) ↑ I.C.T.: Papilledema.

## 3) 3<sup>rd</sup> - 4<sup>th</sup> - 6<sup>th</sup> nerve palsy causing: Paralytic squint & diplopia.

## 4) Gaze palsies:

a) Horizontal gaze palsy (Frontal lobe tumor).

b) Vertical gaze palsy (Brain stem injuries).

## 5) Carotid-cavernous fistula: Pulsatile proptosis.

## 6) Cavernous sinus thrombosis:

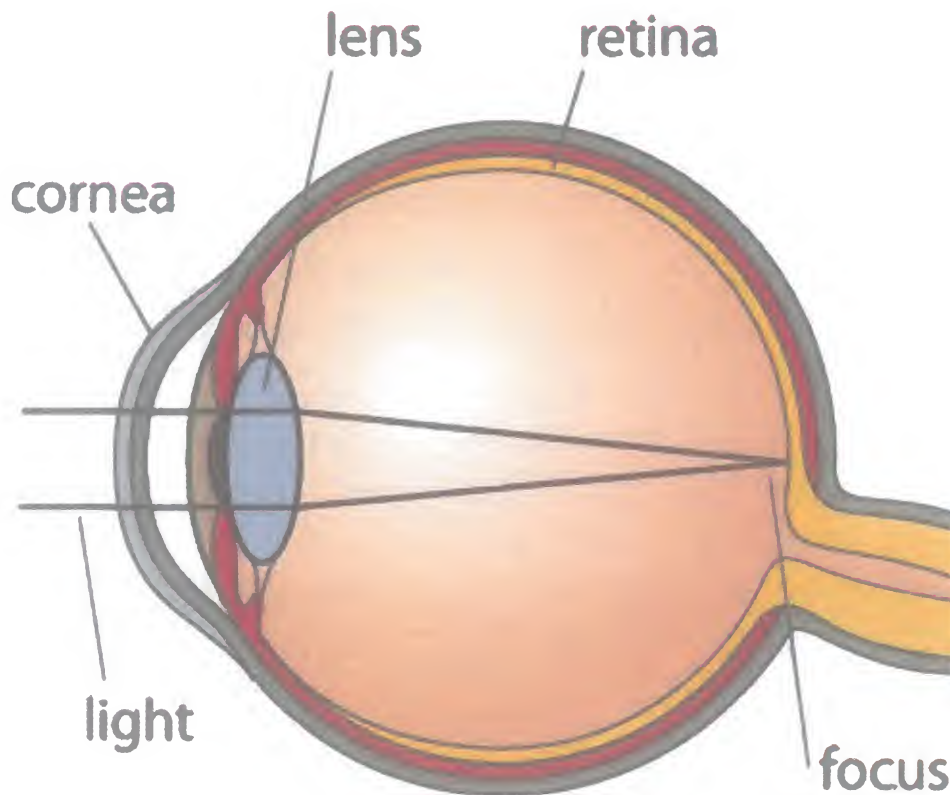
a) Proptosis.

b) Limitation of ocular motility.

## 7) Orbital cellulites (fracture of Para-nasal sinuses).

## 8) Sub-conjunctival hge (orbital vessel injury).

# Errors of Refraction



Elizabeth Morales

# Refraction of the eye

## Tips:

### 1) Types of spherical lenses:

#### a) Convex lenses:

- ❖ Biconvex.
- ❖ Plano convex.
- ❖ Concave-convex.

#### b) Concave lenses:

- ❖ Biconcave.
- ❖ Plano concave.
- ❖ Concave-convex lens.

### 2) Rays when passes through 2 different medias undergo → Refraction.

#### a) Media (1) air.

#### b) Media (2) water.

#### c) $RI \text{ of water} = \frac{\text{Velocity of ray in media (1)}}{\text{Velocity of ray in media (2)}}$

## N.B.1:

1) Convex lens is a (+ lens) while concave lens is (- lens).

2) Power of lens =  $\frac{1}{(\text{Focal distance})}$

3) The eye has two main refractive surfaces: (57D) → Cornea (P = 42D) – Lens (P = 15D)

4) Factor affecting power of lens: Curvature – Index.

5) Factors affecting the eye as an optical system:

- a) Axial length: antero-posterior axis which is normally = 24 mm.
- b) Power of refractive surface.

## N.B.2:

1) Rays coming from a distance **6 meters** or more will fall on the cornea Parallel

2) Rays coming from a distance **less than 6 meters or more** will fall on the cornea divergent.

**Emmetropia:** Is the state of refraction of the eye in which, with accommodation completely relaxed, parallel rays come to a focus on the retina. Rays come out from the retina leave the eye parallel i.e. they meet at infinity,

Infinity is the far point of the emmetropic eye. The retina and infinity are called conjugate foci.

**Ametropia:** Is the state of refraction of the eye in which, with accommodation completely relaxed, parallel rays don't come to a focus on the retina. It is the presence of an error of refraction.



# Myopia (short sightedness)

**Definition:** It is condition of refraction in which with accommodation at rest

- 1) Incident parallel rays come to focus at a point in front of retina.
- 2) Rays emerging from a point retina leave eye convergent & meet at a point in front of eye (punctum remotum).
- 3) Retina and far point are conjugate foci.

## **Etiology:**

1) **Axial myopia (commonest type):** Due to increase in the axial length of the eye & may be:

- a) Simple.
- b) Degenerative.
- c) Congenital.

2) **Refractive myopia:** Due to increase refractive power of the eye, it includes:

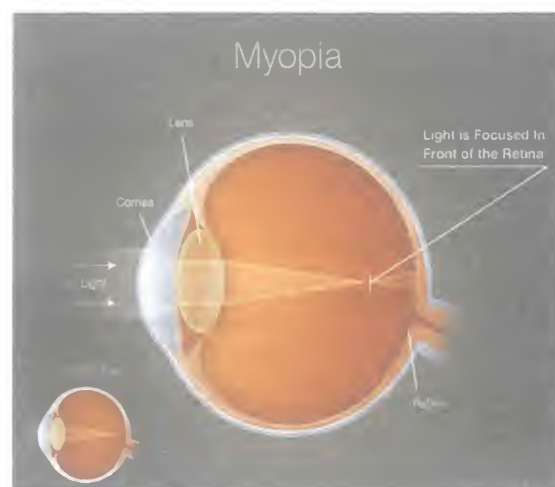
a) **Curvature myopia due to:**

- Increase curvature of the cornea (keratoconus).
- Increase curvature of the lens (lenticonus).

b) **Index myopia as in:**

- Increase R.I. of the nucleus (nuclear cataract).
- Decrease R.I. of the cortex (uncontrolled D.M.).

c) **Anterior displacement of the lens.**



## **Types of axial myopia:**

	Simple	Degenerative (malignant) "Progressive"	Congenital
1) Onset	Around 14 years	Around 7 years	Since birth
2) Progress till	25 years	After 25 years	Stationary
3) Degree	Less than (-8 D)	(-15 D) – (-25 D)	-10 D
4) Degenerative changes	Absent	Present	Less

## **Clinical picture:**

### **Symptoms:**

- 1) **Mascler athenopia:** due to screwing of eyelids to stimulate a pinhole which increases the depth of focus.
- 2) **Simple:** indistinct (unclear) far vision.
- 3) **Malignant:**
  - a) Indistinct far vision.
  - b) Discomfort after near work due to: disproportion between accommodation & convergence.
  - c) Musca volitans: due to vitreous degeneration.
  - d) Retinal symptoms: as:
    - **Photopsia:** flashes due to stimulation of rods & cones by traction of vitreous on the retina.
    - **Night blindness:** due to degeneration of retinal periphery.

### **Signs:**

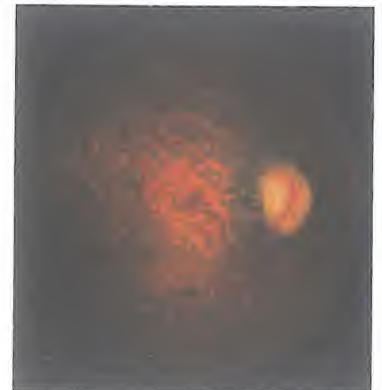
- 1) **Simple:** normal eye.
- 2) **Malignant:**
  - a) **Large eyeball: (pseudo-proptosis):**
    - ❖ Large corneal diameter.
    - ❖ Blue (thin) sclera.
    - ❖ Deep A.C.
    - ❖ Large pupil.
  - b) **Fundus:** Tigroid fundus & signs of complications.
  - c) **Retinoscopy.**
  - d) **Apparent convergent squint.**

## **Complications:**

- 1) **Squint:** (divergent squint) exophoria or exotropia
- 2) **Complicated cataract - lens subluxation.**
- 3) **Retinal changes:**

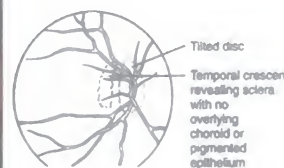
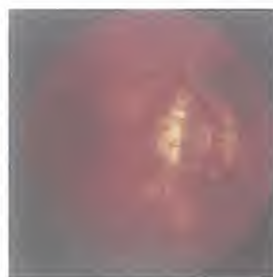
#### a) **Tigroid fundus:**

- ❖ It is red areas (choroidal vessels) alternating with dark areas
- ❖ **Site:** all over the fundus
- ❖ **Cause:** attenuated RPE (Retinal pigment epithelium).



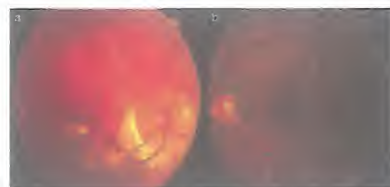
**b) Temporal crescent:**

- ❖ **White crescent.**
- ❖ **Site:** temporal to disc.
- ❖ **Cause:** separation of the choroid from temporal edge of the disc (showing sclera).

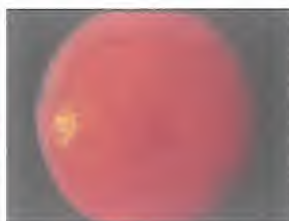


**c) Chorio-retinal degeneration: (scars & atrophic patches):**

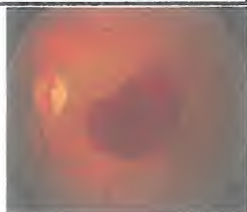
- ❖ It is white areas surrounded by pigmentation.
- ❖ (It may tear → R.D.).



**d) Macular hole.**



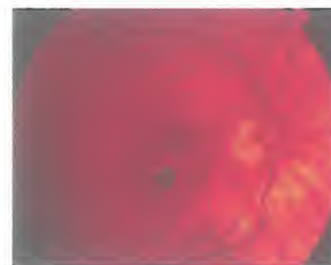
**e) Macular Hemorrhage.**



**f) Fuch's spot: (macular scar)**



- ❖ It is a dark foveal spot.
- ❖ **Cause:** unknown (may be organized Hge).



**g) Lacquer cracks: Breaks in Bruch's membrane of the macular area.**

**h) Peripheral chorioretinal degeneration, retina breaks and retinal detachment.**

**4) Vitreous degeneration (floaters):**

The vitreous becomes **liquefied** & contains **opacities** (musca volitans).

**5) Posterior staphyloma: (only in high myopia):**

- ❖ It is **ectasia** of sclera temporal to the disc.
- ❖ **Site:** temporal to disc.
- ❖ **Detected during:** fundus examination by ophthalmoscope or by U/S.



**Treatment:**

**1) Optical treatment:**

**a) Glasses (concave spherical lens or minus lenses):**

- ❖ Give least minus the makes the patient see  $\frac{6}{6}$  to avoid accommodation.
- ❖ Children give full correction (to allow normal mental development).



❖ Adult:

- **Simple axial** give full correction.
- **progressive (high)** give gradual increase in lens power OR pair of glasses one for far and other for near

**b) Contact Lenses: Advantages:**

- Cosmetically better.
- No significant ↓ in retinal image size.
- Bigger field.

**2) Surgical treatment (refractive surgery):**

**1) Corneal surgery: Refractive myopic surgery:**

**a) Radial keratotomy:**

- ❖ **8-16 incisions** (deep & radial) from the edge of the optical zone to the limbus.
- ❖ **Idea:** the periphery of cornea will bulge & center flattens.
- ❖ **Limits:** degree not less than (- 20 D) & not more than (- 7 D).
- ❖ **Age:** not before the age of 20 years.
- ❖ **Indication:** Patients who is not satisfied with glasses & can't tolerate contact lenses.

**b) Photorefractive excimer laser surgery:**

❖ **Excimer laser keratectomy = photorefractive keratectomy (PRK):**

- Using Eximer laser ablation of the anterior corneal surface.
- Corrects up to (- 10 D).

❖ **Excimer laser intra-stromal Kerato-mileusis (LASIK):**

- A corneal flap is made using a special micro-keratome.
- Eximer laser is directed to the center of cornea, removing part of corneal thickness (stroma).
- Correct up to (-10 D).

❖ **Excimer laser sub-epithelial Kerato-mileusis (LASEK).**

**2) intraocular surgery:**

- a) Phakic IOL.
- b) Clear lens extraction and IOL implantation.
- c) Clear lens extraction only (for - 25).

**N.B. Oral only:**

L	A	S	E	R
Light	Amplification by	Stimulation	Emission of	Radiation

# Hypermetropia

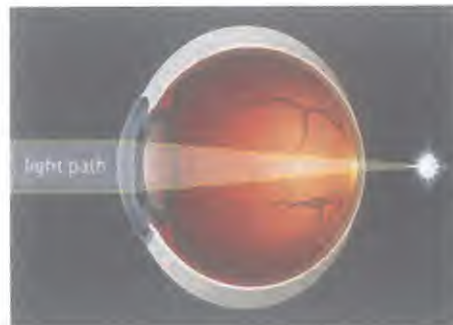
**Definition:** It is a condition of refraction in which with accommodation at rest

- 1) Incident parallel rays come to focus in a point behind the retina.
- 2) Rays emerging from a point on the retina leave the eye divergent & appear as if coming from a virtual point behind the retina (P. remotum).
- 3) Retina and the far point on conjugate foci.

## **Etiology:**

### 1) Axial Hypermetropia:

- a) Due to **small antero-posterior axis (small eye)**
- b) Seen in **children (below the age of 7 years)**.
- c) As the eyes grow they become less hypermetropic:
  - If the eyes reach the proper length (24 mm) → **emmetrope**.
  - If the eyes don't reach the proper length → **remain hypermetrope**.
  - If the eyes exceed the proper length → **become myopic**.
- d) It may be acquired (later on) if the retina is pushed forward as in (**central serous retinopathy**).



### 2) Refractive Hypermetropia: due to ↓ refractive power of the eye.

- a) **Curvature H.** due to flattening of the cornea (cornea plana).
- b) **Index H.** due to ↑ R.I of the lens cortex (immature senile cortical cat. & hypoglycemia).

### 3) Aphakia & posterior lens dislocation.

## **Components of Hypermetropia:**

- 1) **Total hyperopia: (+8 D) =** whole amount of hyperopia under effect of atropine (Cycloplegic), cyclopentolate or tropicamide.

**(i.e. with the tone of ciliary muscles lost)**

- 2) **Latent: (+1 D) =** Amount of hyperopia corrected by normal tone of ciliary muscle.

- 3) **Manifest: (+7 D) =** All amount of hyperopia without effect of atropine = **(7 D) → (total - latent)**.

**N.B.1: (After the normal tone of ciliary muscle restored)**

**N.B.2: Manifest H. corresponds to the highest plus lens giving the maximum visual acuity.**

- a) **Facultative (+3 D) =** Part of manifest which is over corrected by accommodation in children is up to **(14 D)**.

- b) **Absolute (+4 D) =** Part of manifest which is can't be corrected by accommodation.

**N.B.: 1) Hypermetrope accommodate for far object & more accommodation for near object**

**2) After 60 years, all Hypermetropia become absolute (no accommodation)**

## **Clinical picture:**

### **Symptoms:**

1) **Children:** No symptoms (accommodation is strong & correct all H)

All manifest is facultative → no absolute.

2) **Adult:**

a) The patient finds **difficulty for near vision & later for far.**

b) **Accommodative athenopia:**

(Pain in around eye - redness of lid margin - lacrimation - recurrent sty & frequent blinking).

3) **Old age:** difficulty for near and far vision.

### **Signs:**

❖ **Mild degree:** Normal eye.

❖ **High degree:**

1) **Small eye:**

❖ Small cornea.

❖ Shallow A.C.

❖ Small pupil.

2) **Fundus:** bright red reflex, tortuous retinal vessels, pseudo-papillitis

3) **Apparent divergent squint.**

## **Complications:**

1) **Squint:**

a) **Mild:** Latent convergent squint.

b) **High degree:** Manifest convergent squint.

2) **Angle closure glaucoma.**

## **Treatment:**

a) **glasses (convex lens):**

❖ **Children:** full correction to avoid squint.

❖ **Adult:** Highest tolerable convex lens (as patient can't tolerate full correction because they can't relax ciliary muscle at one time due to its spasm)

➤ After 6 months try to increase power of glasses to reach full correction if possible.

❖ **Old age ( pair of glasses):**

1) **For far** → full correction (no accommodation).

2) **For near** → full correction + 3 diopters.

b) **Refractive surgery in high degrees:** IOL implantation & refractive keratoplasty.



# Astigmatism

**Definition:** It's the type of refraction of the eye in which with accommodation at the rest **incident parallel rays do not form a point focus on retina instead, rays will be focused at multiple focal point (line)** due to that the eye has different powers in different meridian.

**Etiology:** Due to irregularities in curvature of cornea or lens:

- 1) **Corneal astigmatism (most of cases):** keratoconus, opacity.
- 2) **Lenticular astigmatism:** lenticonus, subluxation.

**Types of corneal astigmatism:**

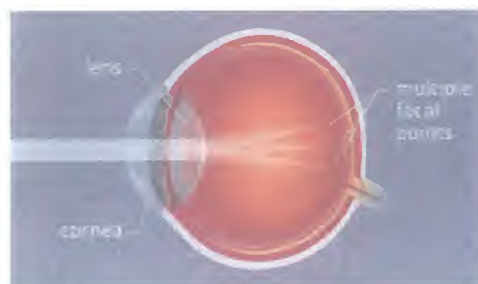
1) **Regular:** It is a hereditary disease.

a) **Rules of regular astigmatism:**

- ❖ 2 major meridians are perpendicular to each other.
- ❖ Transmission between 2 meridians is gradual.

b) **Sub -Rules of Regular Astigmatism:**

- ❖ There are 2 types of regular astigmatism:
  - a) Oblique.
  - b) Straight.
- ❖ In cases of straight regular astigmatism if the vertical meridian is more powerful than the horizontal meridian then it's known as astigmatism with the Rule.



So we have:

1) **Simple astigmatism:** one meridian emmetrope & other ammetrope.

a) **Simple Myopic astigmatism:**

b) **Simple Hypermetropic astigmatism.**

2) **Compound astigmatism:** both meridians are ammetropic but of same type.

a) **Compound myopic astigmatism.**

b) **Compound Hypermetropic astigmatism.**

3) **Mixed astigmatism:** one meridian is myopic & other is hypermetropic.

2) **Irregular:**

- ❖ Meridians of highest & least power are not perpendicular to each other.
- ❖ Transmission from the highest to the least is not regular.
- ❖ e.g.: Corneal Opacity ---- Keratoconus.

### **Clinical picture:**

#### **Symptoms:**

- 1) Accommodative athenopia due to subsequent contractions relaxation of ciliary muscle.
- 2) Blurred vision.
- 3) Distorted object (Difference in curvature so circle appears oval).
- 4) Head tilt.

#### **Signs:**

- 1) **Gross signs:** e.g. keratoconus, corneal opacities, subluxation.
- 2) **Some opening of rings** in the same line is not seen (in Landolt's chart).
- 3) **Astigmatic fan:** some lines appear sharp black other are blurred & grey.
- 4) **Placcido's disc:** shows irregular circles in cases of irregular astigmatism.
- 5) **Keratometer:** the curvature (power) can be measured.
- 6) **Ophthalmoscope:** the op. disc appears oval.
- 7) **Retinoscopy:** by which we can measure power of each meridian.
- 8) **Auto-refractometer.**

### **Treatment:**

- 1) **Regular astigmatism:** Corrected by:

- 1) **Glasses (cylindrical, sphero-cylindrical).**

- a) **Simple:** give cylindrical lenses with its axis perpendicular to meridian to be corrected.
    - b) **Compound or mixed:** give sphero-cylindrical lenses.

- 2) **Soft tonic contact lenses or rigid contact lenses** but less tolerable.

- 3) **Refractive surgery:** astigmatic keratotomy – LASIK.

- 2) **Irregular astigmatism:**

- ❖ **Corneal:** Rigid C.L - keratoplasty.
- ❖ **Lenticular:** Remove lens.

## Anisometropia

**Definition:** A condition of refraction in which difference in refractive power between both eyes more than **4 diopters**.

### **Etiology:**

- 1) **Congenital.**
- 2) **Acquired:** e.g. Unilateral – aphakia.

### Clinical picture:

- 1) **Athenopia.**
- 2) **Loss of binocular vision** (no stereopsis) which may lead to squint.

**Treatment:** Contact lens, IOL & refractive surgery (epi-kerato-phakia).

**N.B.** Can't be treated with glasses as it → Anisokonia → Binocular diplopia.

## Aphakia

**Definition:** It means absence of lens (a = No, phakia = lens)

### Etiology:

- 1) **Congenital:** rare
- 2) **Acquired:**
  - a) **Surgical removal (most common)** after cataract extraction (opaque lens) **OR** after (Subluxation or dislocation) **OR** in high myopia (clear lens extraction = refractive).
  - b) **Posterior dislocation of lens** (same picture of aphakia).
  - c) **Ocular trauma in children** (with opened capsule → absorption of lens matter)

### Clinical picture:

**Symptoms:** Defective vision especially for near (no accommodation).

### Signs:

- 1) **(+ ve) history.**
- 2) **Scar of operation.**
- 3) **A.C.:** deep.
- 4) **Iris:** iridectomy – tremulous (lack of support).
- 5) **Pupil:** jet black – may be round.
- 6) **Absence of 2 out of 3 Perkinji images.**
- 7) **Refraction shift "toward" Hypermetropia.**

### D.D. from post dislocation:

- 1) No history of surgery but + ve history of trauma.
- 2) No scar of previous operation.
- 3) Lens seen in fundus by ophthalmoscope.



## **Optical condition after lens extraction:**

### 1) Loss of accommodation.

### 2) Loss of refractive surface (Refraction toward hypermetropia):

- ❖ A previously emmetrope patient → **Hypermetropia (+10 D).**
- ❖ A previously hypermetropia patient → **Hypermetropia more than (+10 D).**
- ❖ A previously myopic patient → **Hypermetropia less than (+10 D).**
- ❖ Previously myope (- 25.0 D) → **Become emmetrope.**

### 3) Astigmatism against the rule:

- ❖ About 2 diopters (due to: flattening of vertical meridian by scar of operation at upper limbus)

**N.B.: Aphakic correction =  $\frac{2}{3}$  original refraction before op. + 10D.**

## **Treatment:**

### 1) Bilateral aphakia:

- ❖ **Can be corrected by:** glasses – contact lenses – IOL.
- ❖ **Disadvantage of glasses:**
  - Magnified image 25 % - 30 %.
  - Prismatic effect.
  - Aberrations.
  - Limited field.

### 2) Unilateral aphakia: Can be corrected by (contact lenses or IOL) but not glasses to avoid anisokonia and Diplopia.

## **Presbyopia**

**Definition:** It is physiological recession of near point (Punctum Proximum) due to ↓ in accommodation power making near work uncomfortable.

**Etiology:** Due to lens sclerosis as age advances → ↓ its elasticity → ↓ accommodation power.

## **Clinical picture:**

- 1) **Difficulty in near work.**
- 2) **The patient holds the book at a greater distance.**
- 3) **Accommodative Asthenopia → Headache.**

**N.B.: The onset of presbyopia varies with the refractive state of eye:**

❖ **Emmetrope:** around age of 45 ys.

❖ **Hypermetropia:** before that age.

❖ **Myope:** after 45 ys.

### **Treatment:**

- 1) **Reading glasses:** (unifocal, bifocal & multifocal lenses): Plus lenses (added to the far correction) are used to compensate for the lost automatic focusing power of the crystalline lens.
- 2) **Bifocal & Multifocal C.L.:** Limited success.
- 3) **Multifocal IOL:** Used for cataract extraction.
- 4) **Surgery:** Incisions & implants for presbyopia (under research).

## **Contact lenses**

- ❖ Very thin lenses applied directly to the cornea.
- ❖ Principle: Abolishes cornea as a refractive surface replaces it by "Contact lens – fluid lens system".

### **Indications:**

- 1) **Optical:** High errors - anisometropia - irregular astigmatism.
- 2) **Therapeutic:** Neuro paralytic keratitis – resistant ulcers – keratitis metaherptica – wound leaks of small perforation.
- 3) **Cosmetic colored C.L., corneal scar or albinism occupational.**

### **Types:**

- 1) Hard.
- 2) Rigid.
- 3) Soft.

### **Advantages:**

- 1) Large field: No frame + moves with the eye.
- 2) No Anisokonia + no binocular diplopia.
- 3) Cosmetically better.
- 4) Avoid aberrations which result from prismatic effect of lens periphery.

### **Disadvantages:**

- ❖ **Intolerance:** especially with hard C.L.
- ❖ **Lens infection & deposits:** if not properly cleaned.
- ❖ **Corneal abrasion or keratitis.**
- ❖ **Giant papillary conjunctivitis.**
- ❖ **Cornea edema, hypoxia & vascularization.**

# Athenopia

**Defention:** Athenopia or eyestrain is a group of symptoms noticed with visual tasks chiefly after close work, especially in the evening by artificial illumination.

**Symptoms:**

- |   |  |
|---|--|
| 1) Eye ache and burn                          | 4) Hyperemia of the conjunctiva and lid margin |
| 2) Dry sensation leading to frequent blinking |  |
| 3) Lacrimation                                | 5) Headache: usually frontal                   |

**Causes:**

1) Accommodative athenopia:

- |                  |                  |
|------------------|------------------|
| a) Hypermetropia | c) Presbyopia    |
| b) Astigmatism   | d) Anisometropia |

2) Muscular athenopia: due to disproportion between convergence and accommodation as in heterophoria.

# Accommodation

**Definition:** It is the ability of the eye to change its refractive power so that it can focus objects at different distances.

**Mechanism:** contraction of the ciliary muscle results in thickening and increase curvature of the lens, probably due to relaxation of the lens zonule allowing the capsule to compress the lens substance.

- This increase curvature of the lens increases its refractive power allowing the eye to focus on near objects.

**Near point:**

- ❖ The nearest point at which small objects can be clearly distinguished is called near point or punctum proximum.
- ❖ At this point accommodation is the exerted to its maximum.
- ❖ Accommodation is strongest in early childhood and decreases with age.
- ❖ It may be found to be weak or lost in some condition as:
  - 1) Presbyopia
  - 2) Cycloplegic drug as parasympatholytic as atropine, cyclopentolate, tropicamide
  - 3) Third nerve palsy
  - 4) Lens subluxation and aphakia.



**Pediatric**

**ophthalmology &**

**Strabismus**

# Non-Strabismic Pediatric Ophthalmology

## Anatomical Considerations:

- ❖ The axial length at birth is 66% of adult eye. Eye enlarges till 2 years of age.
- ❖ Infants have variable degrees of astigmatism.
- ❖ Majority of children is hyperopic, increases up to 7 years of age, and then diminishes.
- ❖ Eye color darkens over first year of life.
- ❖ Poorly developed dilator pupillae muscle.
- ❖ Fovea matures during first few months of life.
- ❖ Myelination of optic nerve is completed shortly after birth.

## Visual Milestones:

- ❖ 6 weeks: Can fix and follow light source.
- ❖ 3 months: Can fix and follow a slow target and converge.
- ❖ 6 M: Reaches out for objects accurately.
- ❖ 2 years: Picture matching.
- ❖ 5 years: Snellen's chart matching or naming.

## Pediatric Ophthalmic Examination:

It may be done in office or under anesthesia depending on the age of the child and kind of examination (e.g. gonioscopy and tonometry usually require anesthesia).

## Vision Testing in children:

- ❖ Newborn: Evaluation of the red reflex and pupillary testing.
- ❖ Infants below one year: opto-kinetic nystagmus and electrophysiological study (as VEP).
- ❖ 6 months → 2 years: preferential looking.
- ❖ 2 – 4 years: picture naming test.
- ❖ 4 – 5 years: Adult acuity tests as Snellen's chart.

## Indications for optical correction in children:

- ❖ The visual demands of the child must be taken into account,  
e.g.: An 8 months child having (1 D) myopia will not benefit from optical correction, while for a third grade student the benefit suddenly becomes remarkable.
- ❖ In amblyopia age group (age 6 years or younger):
  - All significant ametropia should be corrected.
  - This includes a myopia or astigmatism > 2.0 D and hyperopia > 4.0 D.
- ❖ Contact lens use or even laser correction may be a solution for high anisometropia with significant amblyopia.

# The extra-ocular Muscles

- ❖ There are 6 EOM (4 recti & 2 oblique) which rotate the eye around:
- ❖ 3 axes:
  - Vertical axis → adduction and abduction.
  - Horizontal axis → elevation and depression.
  - Antero-posterior axis → intorsion and extorsion.
- ❖ When the two eyes are directed straight ahead, this called (primary position) and in this position no muscle is contracting.

## Rectus muscles:

- ❖ Origin: arise from a fibrous ring placed around the optic foramen and medial part of SOF → annulus of Zinn.
- ❖ Insertion: to the sclera at a variable distances from the limbus.  
(Medial rectus 5.5 mm, inferior rectus 6.5 mm, lateral rectus 6.9 mm, superior rectus 7.7 mm)

## Oblique muscles:

- ❖ Origin:
  - **Superior oblique**: from the orbital apex above and medial to the annulus of Zinn.
  - **Inferior oblique**: from the orbital floor lateral to the opening of NLD.
- ❖ Insertion:
  - ❖ **Superior oblique**: the muscle passes forward and medially then becomes tendinous and passes through a fibrous ring at the antero-medial part of the orbital roof near the orbital margin (trochlea) → reflected backward and laterally inserted in sclera in the upper postero-lateral quadrant.
  - ❖ **Inferior oblique**: the muscle passes backward and laterally below the globe to be inserted in the sclera in the lower postero-lateral quadrants (5 mm from optic nerve almost over the macula).

## Nerve supply:

- ❖ 3<sup>rd</sup> n. (oculomotor): Superior rectus, medial rectus, inferior rectus, Inferior oblique.
- ❖ 4<sup>th</sup> n. (trochlear): Superior oblique.
- ❖ 6<sup>th</sup> n. (abducent): Lateral rectus.

**Blood supply:** Muscular branches of ophthalmic artery.



### Action:

- 1) Medial rectus: adduction.
- 2) Lateral rectus: abduction.
- 3) Superior rectus: **Primary action:** elevation, **subsidiary action:** adduction & intorsion.
- 4) Inferior rectus: **Primary action:** depression, **subsidiary action:** adduction & extorsion.
- 5) Superior oblique: **Primary action:** intorsion, **subsidiary action:** depression & abduction.
- 6) Inferior oblique: **Primary action:** extorsion, **subsidiary action:** elevation & abduction.

❖ Superior muscles are intorters while inferior muscles are extorters.

❖ Recti are adductors while obliques are abductors.

❖ Antagonistic muscles: LR & MR of the same eye.

❖ Synergistic muscles : SR and IO of same eye

❖ Looking direct upwards by (IO + SR).

❖ Looking direct downwards by (SO + IR).

❖ On abduction → Recti are only elevator and depressor.

❖ On adduction → Obliques are only elevator and depressor.

**The muscle axis:** It is the line between the origin and insertion.

### The muscle action is:

- ❖ Maximum if muscle axis is parallel to anatomical axis.
- ❖ Minimum if muscle axis is perpendicular to anatomical axis.

**N.B.: Remember the six cardinal directions of gaze. (Clinical sheet)**

## Tips

### 1) Mono-ocular movement: (duction):

- |               |              |
|---------------|--------------|
| a) Adduction  | b) Abduction |
| c) Elevation  | e) Extorsion |
| d) Depression | f) Intorsion |

❖ (Sherrington's law): Excitatory impulse flows to agonist and inhibitory to antagonist.

### 2) Binocular eye movements:

- ❖ Vergence → both eyes move against each other → Convergence – Divergence.
- ❖ Versions → binocular movement in the same direction.

### Two laws for version:

#### 1) Yoke muscle:

- ❖ Pair of muscles one in each eye able to move both eyes in the same direction ( version )

#### 2) Herring law: Equal and simultaneous impulse flows to yoke muscle.

# Squint (strabismus)

**Optic axis:** The line connecting center of the cornea, lens, retina (post pole).

**Visual axis:** The line connects fovea & object of fixation passing through the nodal point.

**Angle alpha:** The angle between optic & visual axis.

❖ In emmetrope: (+5)

❖ In hypermetrope: more than (+5) → apparent divergent squint.

❖ In myope: less than (+5) or even negative → apparent convergent Squint.

**Orthophoria:** When visual axis's of both eyes are directed (or coincide) at object of the regard.

**Definition of squint:** A condition in which the visual axes of the 2 eyes are "not" directed to the same object.

## Types:

1) Apparent squint.

2) True squint:

a) Latent squint.

b) Manifest squint:

➤ Paralytic.

➤ Concomitant.

## Diplopia:

**Definition:** Simultaneous perception of two images for the same object by both eyes.

**Cause of Binocular diplopia:** Stimulation of Non correspond retinal points.

# Apparent Squint

**Definition:** A condition in which the person appears to have squint although the 2 visual axes are directed to the same object.

## Etiology:

1) Apparent convergent squint:

a) **Epicanthus:** a skin fold over the medial canthus (congenital, racial).

b) **High myopia:** due to (-ve) angle alpha.

c) **Small IPD:** (normal IPD: 55 – 70 mm).

2) Apparent divergent squint:

a) **Lateral ankyloblepharon:** adhesion between the lid margins. →

b) **High Hypermetropia:** due to large (+ve) angle alpha.

c) **Large IPD** (hypertolerism = wide distance between the 2 eyes).



3) Apparent upward deviation: Ptosis.

4) Apparent downward deviation: Lid retraction.

**Diagnosis:**

1) Presence of a cause.

2) Cover test → no movement.

3) Corneal light reflex: normally centered.

**Treatment:** Treatment of the cause only (as there is no squint).

## Latent squint (Heterophoria)

**Definition:** It is tendency of the eye to deviate them orthoporic position but this tendency is corrected by brain to maintain single binocular vision.

**Etiology:**

**EOM imbalance:**

1) Uncorrected errors of refraction: Leading to disproportion between accommodation and convergence.

❖ **Hypermetropia:**

➤ Increased accommodation:

➤ Latent convergent squint.

➤ Increased convergence.

❖ **Myopia:** Latent divergent squint.

2) Mild weakness of one or more of EOM, not sufficient to produce manifest squint.

**Types:**

1) **Exophoria**: the eye tends to deviate outwards

2) **Esophoria**: the eye tends to deviate inwards.

3) **Hyperphoria**: the eye tends to deviate upwards.

4) **Hypophoria**: the eye tends to deviate downwards.

**Symptoms:** (latent squint is present in large percentage of population).

1) Compensated cases: Show no symptoms (most cases).

2) Decompensated cases: may show:

a) **Symptoms due to trial, to maintain binocular vision** → muscular athenopia (eye strain).

After prolonged close work, the eyes are red with:

❖ Ocular pain - Headache - Lacrimation - Recurrent stye & blepharitis.

b) **Symptoms due to failure to maintain binocular vision** (Latent → manifest) (lost interest for fixation or fatigue)

➤ Running letters or double image due to intermittent diplopia.



**Diagnosis: Principle:** depends on abolishing the binocular vision (B.V.) by covering one eye therefore presenting different images to each eye, so the brain will lose interest of B.V. and then latent squint will change to manifest squint.

**1) Cover-uncover test:** The idea is to abolish the stimulus for binocular single vision by covering one eye (cover test):

- a) Ask the patient to fix an object.
- b) Each eye is covered separately.
- c) The occlude is quickly removed and the examiner notes whether or not the eye under cover had deviated. The examiner must also note if the eye makes a movement inwards or outwards to pick up fixation once cover is removed
- d) If the covered eye deviates under the cover and when the cover is removed returns to the original fixing position, **latent squint is present**.

**2) Maddox rod (for far):**

- a) Maddox rod is a group of red cylinders arranged parallel to each other.
- b) They produce a red line image from a point source of light  
(This red line is perpendicular to the axis of the cylinders).
- c) The rod is placed in front of one eye and the patient fixates a source of light at a distance of **6 meters**.
- d) An orthophoric (normal) sees the point of light coinciding with the middle of the red line.



**3) Maddox wing:** (for near):

- a) The Rt. Eye sees 2 arrows (vertical & Horizontal).
- b) The Lt. Eye sees 2 scales (V. & H.).
- c) An orthophoric sees the arrows points to the zero.
- d) In heterophoria the arrows either the horizontal or the vertical one point outside zero (the patient can read directly his angle of squint).



### **Treatment:**

**1) Compensated cases:** no symptoms → No Treatment.

**2) Decompensated cases:**

- a) Correct any error of refraction.
- b) Strengthen the weak muscle by:
  - **Orthoptic exercises:** especially in convergence insufficiency.
  - **Exercising prisms:** increase muscular effort.
  - **Surgery:** if all the above failed (in large degrees).
- c) Relieving prisms: Disadvantages → Latent change Manifest.

Exercising prisms  
(Base toward deviation)  
Relieving prisms  
(Apex toward deviation)

# Paralytic Squint

**Definition:** It is manifest squint due to paralysis of one or more of EOM.

**Etiology:** It is due to: lesion in the muscles or its nerve supply

(Lower motor neuron lesion: nuclear, nerve or muscle lesion).

## 1) Nuclear lesions:

- ❖ **Congenital:** e.g. Absence of the nucleus.
- ❖ **Inflammatory:** e.g. encephalitis.
- ❖ **Vascular:** e.g., thrombosis, embolism & Hemorrhage.
- ❖ **Neoplastic:** tumors of brain stem (Midbrains, pons).
- ❖ **Other (toxic):** as in diphtheria and with alcohol, lead.

## 2) Nerve lesions:

- ❖ **Congenital.**
- ❖ **Inflammatory:** meningitis, cav. Sinus thrombosis.
- ❖ **Vascular:** Hge (subdural, subarachnoid) & cavernous sinus Thrombosis.
- ❖ **Neoplastic:** Brain tumor, orbital Tumor.
- ❖ **Trauma:** e.g., fracture base of the skull.
- ❖ **Metabolic:** DM

## 3) Muscle lesion:

- ❖ **Congenital:** e.g. maldevelopment of the muscle (hypoplasia).
- ❖ **Trauma:** Contusion or Hge in muscle Sheath or fracture orbital bones.
- ❖ **Neuro-muscular:** Myopathy & Myasthenia gravis.
- ❖ **Dysthyroid ophthalmopathy (thyroid eye disease).**
- ❖ **Neoplastic:** Orbital tumors.

### **N.B.:**

#### ❖ Ophthalmoplegia:

1) External ophthalmoplegia → paralysis of EOM.

2) Total ophthalmoplegia → paralysis of EOM and intra OM.

❖ Superior orbital fissure syndrome: it includes affection of 3<sup>rd</sup>, 4<sup>th</sup>, 6<sup>th</sup> and 1<sup>st</sup> division of the 5<sup>th</sup> cranial nerves.

❖ Orbital apex syndrome: It includes SOF syndrome & op. nerve affection.

### **Clinical picture:**

- 1) **Limitation of ocular movement:** In the "same" direction of action of the paralyzed muscle.
- 2) **Manifest squint:** To the "opposite" direction of the action of the paralyzed muscle, (the paralyzed muscle loses its tone → the antagonistic draws the eye toward it).
- 3) **The angle of deviation in paralytic squint changes in the different direction of gaze:**
  - ❖ The 2<sup>nd</sup> angle of deviation > The 1<sup>st</sup> angle of deviation
  - ❖ **Primary angle:** it is the angle detected in the primary position of the gaze (The normal eye is fixing).
  - ❖ **Secondary angle:** it is the angle detected in the secondary position of gaze (side gaze) when the patient is trying to fix by the squinting paralyzed eye while the normal eye is covered.  
(The angle here is increased due to the excessive movement of the sound eye).
- 4) **Diplopia:**
  - a) **Characters:**
    - ❖ Binocular: disappears if one eye is covered.
    - ❖ More in the "same" direction of action of the paralyzed muscle.
    - ❖ It may be crossed or uncrossed.

#### **N.B.:**

- ❖ **Uncrossed (Homonymous) diplopia:** when the false image falls **on the same side of the paralyzed "eye"** e.g., in lateral Rectus paralysis.
- ❖ **Crossed (Heteronymous) diplopia:** when the false image falls **on the opposite side of the paralyzed "eye"** e.g., in medial Rectus paralysis.

#### **b) The patient fights diplopia by:**

- ❖ Suppression (child).
- ❖ Compensatory head position (in the direction of action of paralyzed muscle).
- ❖ Covering one eye.

#### **5) False projection (past-pointing):**

- ❖ It is false estimation or orientation of objects (with the normal eye covered).
- ❖ **Cause:** the brain sends excessive impulses to the paralyzed muscle to force it to contract.



## Complications:

1) **Suppression (in young):** of the false image (to avoid diplopia) → amblyopia  
(Functional diminution of vision).

## 2) **Muscle changes (in old):**

- a) Direct antagonist may enter in contracture (on prolonged palsy).
- b) Over action of the contralateral synergist (due to excessive impulses).
- c) Under action of the contralateral antagonist (due to inhibitory impulses).

## Diagnosis:

- ❖ Testing of ocular motility: ask the patient to fix your finger in.
- ❖ Diplopia chart, the 6 cardinal directions.
- ❖ Hess screen.

## Treatment:

### 1) **Treatment of the cause:**

- ❖ This may lead to **complete recovery** (spontaneous nerve regeneration).
- ❖ During that: **cover one eye** (squinting eye to avoid binocular diplopia).

### 2) **Surgical treatment:** Required if no recovery occurs **after 6 months** of treatment.

- a) If the muscle is weak (the paralyzed eye cross the mid-line i.e. paresis)
  - **Strengthen it (by resection) & weaken its direct antagonist (recession).**
- b) If the muscle is completely paralyzed (cannot move to mid-line) → Muscle transposition  
(Jensen operation only in LR paralysis).

## 3<sup>rd</sup> nerve Palsy:

- ❖ Ptosis (due to levator paralysis).
- ❖ Paralysis of accommodation (paralysis of ciliary muscle).
- ❖ No diplopia (due to ptosis).
- ❖ Dilatation of the pupil (paralysis of constrictor muscle).
- ❖ Proptosis (mild) due to loss of tone of the paralyzed muscle.
- ❖ Paralysis of EOM + deviation out (lat. R.).

## 4<sup>th</sup> nerve palsy:

- ❖ Paralytic squint: the eye deviates up.
- ❖ Diplopia: increase on looking down.

## 6<sup>th</sup> nerve palsy: If the right side is affected:

- ❖ Rt. Cony. Squint (angle of squint is larger on looking to the RT).
- ❖ Limitation of ocular Movement to the RT.
- ❖ Diplopia: uncrossed and increase on looking to the RT.
- ❖ False p. on looking to the RT.
- ❖ Face turn to the RT.

# Concomitant Squint

**Definition:** It is a manifest squint in which the visual axes of the two eyes don't meet at object of regard and they maintain their relation constant in all directions of gaze.  
(1<sup>st</sup> & 2<sup>nd</sup> deviation are equal).

## **Etiology:**

- ❖ **Binocular vision:** is fully developed between the ages of 6 months and 6 years.
- ❖ Any obstacle to binocular vision during this critical period would result in → Ocular deviation.

## **Such obstacles might be:**

### ❖ **Refractive (Uncorrected error of Refraction):**

- In Hypermetropia (more than 3D), the child accommodates to see clear.
- Accommodation is associated with convergence → Esotropia.
- In Myopia, relaxation of accommodation & lack of convergence Exotropia (concomitant divergent squint).

### ❖ **Non -refractive:**

- **Congenital:** Esotropia is more common than exotropia:
  - a) Peripheral motor obstacle: any anomaly of EOM.
  - b) Central obstacle: (defective development of presumed fusion center or visual pathway).
- **Sensory obstacle:** due to monocular impaired vision (unilateral cataract); if the visual acuity in one eye is weak the brain will suppress it → if unilateral squint.

**Sequelae of concomitant squint:** With the onset of squint, the images of the object will fall on non-corresponding points of the retina binocular diplopia must occur. But as the age of the patient is young, the brain finds a solution for this and diplopia doesn't persist.

## **These solutions are:**

### **1) Suppression:**

- ❖ It is active neglect by the brain for the image seen by the squinting eye.
- ❖ If suppression becomes permanent, this is known as **Amblyopia**.

### **2) Eccentric fixation:**

- ❖ It occurs in some cases of amblyopia when the patient develops the ability to fix objects by a part of the retina other than the macula (**false macula**).
- ❖ It is due to **dense suppression of the macula**.
- ❖ Covering fixing eye → **squinting eye fix the object without any mov. with bad V/A ( $\frac{6}{60}$ )**

## **Clinical picture:**

**Symptoms:** Deviation of one or both eyes.

### **Signs:**

1) **Test the ocular motility:** In the 6 cardinal directions → to exclude paralytic squint.

2) **Measure the visual acuity:**

- ❖ If equal in both eyes → alternating squint, (patient can fix by both eyes).
- ❖ Poor in the squinting eye → unilateral squint (Rt. Or Lt.)

3) **The cover test:**

- ❖ Ask the patient to fix an object (1<sup>st</sup> at a distance then at near).
- ❖ **Cover the fixing eye:**
  - The squinting eye will fix.
  - The fixing eye will deviate.
- ❖ **Remove the cover:**
  - If the new position remains → **Alternating Squint (Suppression)**
  - If the original position returns → **unilateral Squint (Amblyopia)**
- ❖ **No movement in:**
  - a) Eccentric fixation (**low vision**).
  - b) Apparent squint (**good vision**).
  - c) Paralytic squint.
  - d) Blind eye. (No PL).

### **N.B.: Benefits of cover test:**

1) **Latent:** Diagnosis of latent squint.

2) **Paralytic:** Proved that Secondary angle squint is larger than angle in cases of paralytic.

3) **Concomitant:** Differentiation between unilateral & alternating squint.

4) **Concomitant:** Diagnosis of eccentric fixation.

4) **Measurement of the angle of squint:**

- **Corneal light reflex (Hirschberg test):** depend on first purkinje image:
  - ❖ Hold a torch in front of the patient's nose.
  - ❖ Observe the light reflex on the cornea of the squinting eye.
  - ❖ Rough test.



➤ **Synaptophore (major amblyoscope):**

- ❖ The synaptophore is an instrument, composed of 2 tubes which can be moved on a graded scale.
- ❖ Ask the patient to look through the tubes.
- ❖ Then ask him to rotate the tubes until the 2 test objects (at the tube end) are superimposed (e.g. the bird inside the cage).
- ❖ The angle between the 2 tubes = angle of squint.



- **Prism cover test** → angle of squint nearly equals prism power that (krimsky test) abolishes the movement on cover test.

**5) Assessment of binocular vision:**

- ❖ **Definition:** It is the coordinated use of the 2 eyes to produce single mental impression.

- ❖ **Values:**

- Stereopsis (depth perception).
- Larger field.
- Optical defects and field defects (scotoma) in one eye are masked by the other.

- ❖ **Grades:**

- **Simultaneous macular perception:** (The ability to perceive and superimpose the images of the 2 maculae).
- **Fusion** (the ability to fuse 2 images with control).
- **Fusing** (2 similar objects with missing details in each → one complete image).
- **Stereopsis** (the ability to perceive the depth).

- ❖ **Assessment:**

- **Synoptophore:**

- **Worth's 4 dot test:**

- ❖ Ask the patient to wear red-green goggles.
- ❖ Then, ask him to look to the colored illuminated dots.

- ❖ **Results:**

- a) **Binocular vision:** 4 dots.
- b) **Diplopia:** 5 dots.
- c) **Left suppression:** 2 red dots.
- d) **Rt. Suppression:** 3 green dots.



### Infantile congenital esotropia:

❖ **Presentation:** within the first 6 months of life.

❖ **Examination reveals:**

- a) Angle of deviation is usually large.
- b) Fixation is usually alternating.
- c) Refractive error is normal for the age of the child (not excessively hypermetropic).

❖ **Management:**

- a) Refractive error and amblyopia treated first.
- b) Eyes are aligned surgically, usually at the age of twelve months.



### Refractive (accommodative) esotropia:

It is associated with activation of the accommodative reflex in response to excessive hypermetropia.

❖ **Presentation:** usually at age 2 - 3 years.

❖ **Examination:**

- a) Visual acuity: to determine if one eye sees worse.
- b) Corneal reflex test.
- c) Cover test.

### Treatment:

❖ **Aim:**

- To build and restore binocular vision.
- To improve and preserve V. acuity in squinting eye.
- To improve the cosmetic appearance.

❖ **Time:** as early as possible (before age of 7 years).

- To avoid amblyopia.
- To allow binocular vision to develop → Cases treated after age of 7 years are treated for cosmetic appearance.

❖ **Lines of treatment:**

- **Atropine eye ointment:** For accommodative squint in children before 2 years till can wear glasses (to relieve accommodation associated with convergence).
- **Glasses:** correction of error of refraction, as soon as the child can wear it.
- **Amblyopia therapy:** by covering the sound eye to improve the vision of the amblyopic eye.

❖ Plan of TTT:

➤ Adult: Surgical correction of squint → for cosmetic purpose only.

➤ Children:

1) **Error not matching:** With type – squint → squint surgery followed by Orthoptic treatment.

2) **Error matching:**

**Glasses:**

❖ If successful → orthoptic treatment.

❖ If Not succession → **Occlusion therapy** → If success full → Orthoptic treatment  
→ Not succession → **Squint surgery**

**N.B.:**

	Concomitant	Paralytic
Ocular motility	Free	Limited
Angle of squint	Constant ( $1^{\text{ry}} = 2^{\text{ry}}$ )	( $2^{\text{ry}} > 1^{\text{ry}}$ )
Diplopia	Absent due to suppression	Present
False projection & Compensatory Heads position	Absent	Present

❖ Surgical treatment:

❖ **Indication:**

- No error
- Residual angle (after glasses and orthoptic training).
- Age > 7 years (for cosmetic appearance).

❖ **Technique:**

1) Weakening op: Recession: carrying the insertion backward.

2) Strengthening op: Resection: shortens the muscle → increase power.

**Rules:**

- ❖ Every 1 mm (recession or resection) of M.R corrects 2 – 3.
- ❖ Every 1 mm (recession or resection) of LR corrects 1.5°.
- ❖ In alternating squint, symmetrical surgery is required.



# Amblyopia

**Definition:** It is unilateral or rarely bilateral decrease in the best corrected visual acuity in the absence of eye pathology or visual pathway disease.

**Etiology and Type:** Main cause is either: Vision deprivation **OR** abnormal binocular interaction.

- 1) **Stimulus deprivation amblyopia:** Caused by media opacity or severe ptosis.
- 2) **Anisometropic amblyopia:** Caused by difference in refractive error between both eyes leading to abnormal binocular interaction.
- 3) **Strabismic amblyopia:** Strabismus leads to abnormal binocular interaction and occurrence of diplopia leading to suppression of the image of the squinting eye.

## **Diagnosis:**

- ❖ **Visual acuity is less in the amblyopia eye** by two lines or more on Landolt's chart in absence of organic lesion.
- ❖ **Neutral density filter:**
  - Will cause significant drop by 2 lines or more in case of organic lesion.
  - No significant drop in V/A in cases of amblyopia.

**Treatment:** Sensitive period during which amblyopia can be reversed up to:

- ❖ 7 to 8 years in **strabismic amblyopia**.
- ❖ 11 years in **anisometropic amblyopia**.

## **By:**

- ❖ **Occlusion therapy.**
- ❖ **Pinnalization** (Atropine eye drops in the sound eye so the child won't see well with this eye).

The eye &

Systemic diseases

❖ The most common systemic diseases affecting the eye **include:**

- Infectious diseases.
- Collagen diseases.
- Metabolic diseases.
- Intoxications.
- Hematological disorders,
- Endocrine disorders,
- Nutritional deficiencies.

## Infectious Diseases

### **Tuberculosis:**

- ❖ Tuberculosis (TB) is a **chronic granulomatous infection caused by tubercle bacillus of Mycobacterium**. Two species responsible for TB in human are the human strain *M. tuberculosis* acquired by inhalation and the bovine strain *M. bovis* which is acquired by drinking unpasteurized milk from infected cattle.
- ❖ TB is primarily a pulmonary disease but it may spread by blood to other sites to form generalized (miliary) infection.
- ❖ **Ocular features:**
  - **Anterior segment manifestations:** as eyelid lesions as reddish brown nodules (lupus vulgaris) or a cold abscess, phlyctenular keratitis, granulomatous anterior uveitis.
  - **Posterior segment manifestations:** may show focal or multifocal choroiditis, occlusive retinal periphlebitis. It may also result in optic neuropathy and ocular motor palsies.
- ❖ It is worth noting that tuberculosis should be differentiated from sarcoidosis.

### **Leprosy:**

- ❖ Leprosy is a chronic granulomatous infection caused by **an intracellular bacillus (mycobacterium leprae)** which has an affinity for skin, peripheral nerves and anterior
- ❖ Transmission is through **the upper respiratory tract**.
- ❖ **Ocular features:** include anterior uveitis, keratitis, madarosis, trichiasis, conjunctivitis and scleritis

### **Acquired syphilis:**

- ❖ Syphilis is caused by a **spirochaete Treponema pallidum**. In adults the disease is usually **sexually acquired** but transmission can also be **by kissing or blood transfusion**.
- ❖ **Ocular features:** are uveitis, interstitial keratitis, madarosis, optic neuritis, Argyll Robertson pupils and ocular motor nerve palsies.
- ❖ Congenital syphilis is transmitted **transplacental** and commonly manifests by anterior uveitis and interstitial keratitis and uncommonly by pigmentary retinopathy.



### **Acquired immune deficiency syndrome (AIDS):**

- ❖ Acquired immune deficiency syndrome (AIDS) is caused by the **human immunodeficiency virus (HIV)**. Mode of transmission is mainly by **sexual intercourse or by contaminated blood in needles, transplacental or via breast milk**. HIV targets (**CD4 + T helper lymphocytes**), which are vital to the initiation of the immune response to pathogens.
- ❖ **Ocular features are mainly those of opportunistic infections and tumors:**
  - **Anterior segment manifestations are:** blepharitis, eyelid Kaposi sarcoma, multiple molluscum lesions and severe herpes zoster ophthalmicus or orbital cellulitis, Conjunctival Kaposi sarcoma, squamous cell carcinoma, herpes simplex and herpes zoster keratitis and anterior uveitis.
  - **Posterior segment may show:** HIV retinopathy, cytomegalovirus retinitis and toxoplasmosis.

### **Congenital rubella:**

- ❖ Congenital rubella (German measles) results from **transplacental transmission of the virus from infected mother**. It may lead to serious chronic fetal infection and malformations.
- ❖ **Ocular features** include cataract, microphthalmos, glaucoma, retinopathy, keratitis, anterior uveitis and severe refractive errors.

### **Toxoplasmosis:**

- ❖ Toxoplasmosis is caused by **toxoplasma gondii protozoan**. Infection occurs by ingestion of undercooked meat or accidental contamination of hands when dispensing cat litter trays or transplacental.
- ❖ **Congenital toxoplasmosis manifests by** bilateral healed chorioretinal scars. Acquired toxoplasmosis is the most frequent cause of infectious retinitis in immunocompetent individuals.

### **Mucormycosis:**

- ❖ Mucormycosis is a **very rare opportunistic infection** caused by **fungi of the family Mucoraceae** which typically affects patients with diabetic ketoacidosis or immunosuppression.
- ❖ This aggressive and potentially fatal infection is acquired by the inhalation of spores which give rise to an upper respiratory infection.
- ❖ The infection then spreads to the contiguous sinus and subsequently to the orbit and brain. Invasion of blood vessels by the hyphae results in **occlusive vasculitis with ischemic infarction of orbital tissues, ophthalmoplegia and retinal vascular occlusion**.

### **Other infectious diseases and ocular presentations:**

- 1) **Keratoconjunctivitis:** this can be a part of many viral infections as measles, chicken pox and rubella. Granulomatous infections as TB and syphilis can produce chronic granulomatous conjunctivitis.
- 2) **Sub-conjunctival haemorrhage:** may occur in hemorrhagic fevers as in rift valley fever, Ebola virus, and spirochaetal diseases.
- 3) **Uveitis:** non-specific uveitis may be a manifestation of many viral infections. Metastatic purulent uveitis (endophthalmitis) can occur with pyogenic bacteria as in intravenous drug abusers. Granulomatous uveitis occurs with TB and syphilis.
- 4) **Retinitis:** specific forms of retinitis may occur with cytomegalovirus infection in AIDS patients. Acute retinal necrosis can occur with herpes simplex retinitis. Congenital rubella can produce a retinitis pigmentosa-like disease in the newly born (salt and pepper fundus). Cysticercosis may give rise to sub-retinal parasitic cysts and toxocara species can produce a retinal granuloma in children.
- 5) **Optic neuritis and subsequently optic atrophy** can occur with encephalitis and meningitis of viral or bacterial origin. Optic atrophy may be a complication of therapy as with use of ethambutol (anti-tuberculous drug).
- 6) **Orbital parasitic cysts:** may be seen in hydatid disease (echinococcus granulosus) and myositis of the extra-ocular muscles is a characteristic feature of Trichenella spiralis infestation.

## **Hematological Diseases**

### **1) Coagulation disorders:**

- ❖ As: hemophilia, thrombocytopenia and anti-coagulant therapy.
- ❖ Can produce: hemorrhage anywhere in the eye the most significant of which are vitreous and retinal hemorrhages.

### **2) Severe anemia:**

- ❖ Can produce: pallor of the conjunctiva, retinal hemorrhages and optic disc edema.
- ❖ Sick-cell anemia can produce retinal arteriolar occlusions and retinal neovascularization.

### **3) Hematological malignancies:**

- ❖ As leukemia and lymphomas can produce a wide-variety of manifestations including sub-conjunctival hemorrhage, orbital infiltrations, proptosis, uveal nodules and retinal pale-centered hemorrhages (Roth spots).

# Collagen Diseases

- ❖ This is a group of diseases involving the joints and connective tissue all-over the body. They include rheumatoid arthritis, polyarteritis nodosa, systemic lupus erythematosus, Sjogren syndrome, and scleroderma.

## Rheumatoid arthritis:

- ❖ It is an autoimmune disease characterized by **symmetrical destructive polyarthropathy**. There is circulating antibodies called rheumatoid factor (RF). It is more predominant in **females**.
- ❖ **Systemic features:** arthritis involving small joints and when advanced involves larger joints, and may result in vasculitis.
- ❖ **Ophthalmic features:** keratoconjunctivitis sicca, scleritis and peripheral ulcerative keratitis.

## Polyarteritis nodosa:

- ❖ It is an idiopathic, collagen vascular disease affecting small and medium sized arteries with frequent aneurysm dilatation. **Males** are more affected.
- ❖ **Systemic features:** purpura, easy bruising and vasculitis.
- ❖ **Ophthalmic features:** Peripheral ulcerative keratitis, scleritis, and occlusive retinal periarteritis.

## Systemic lupus erythematosus (SLE):

- ❖ It is an autoimmune disease with circulating immune complexes with wide spread vasculitis. **Females** are more affected.
- ❖ **Systemic features:** mucocutaneous facial rash, vasculitis, telangiectasia, oral ulceration, arthritis, myositis and tendonitis.  
Renal cardiovascular, pulmonary, neurological and reticuloendothelial affection.
- ❖ **Ophthalmic features:** madarosis, keratoconjunctivitis sicca, peripheral ulcerative keratitis, scleritis and retinal vasculitis.

## Sjogren disease:

- ❖ It is an autoimmune disease characterized by inflammation and destruction of lacrimal gland and salivary glands. **Females** are more affected.
- ❖ **Systemic features:** enlargement of salivary glands, decreased salivary secretion resulting in **fissured tongue, dry nasal passages**.
- ❖ **Ophthalmic features:** keratoconjunctivitis sicca, Adie's pupil.



### **Scleroderma:**

- ❖ It is idiopathic, chronic, Connective tissue disease affecting skin and internal organs.
- ❖ **Systemic features:** tightening and thickening of hands, face and trunk (waxy appearance), Expressionless face.
- ❖ **Ophthalmic features:** keratoconjunctivitis sicca, eyelid tightening.

## Endocrinal disorders

- 1) **Thyroid dysfunction.** (see before)
- 2) **Diabetes mellitus.** (see before)
- 3) **Complicated cataract:** can be seen in cushing syndrome and hypoparathyroidism
- 4) **Optic nerve compression and bitemporal hemianopia** are common manifestations of pituitary chromophobe adenomas.

## Metabolic Diseases

- 1) **Metabolic cataract:** Can be seen in: galactosemia, renal rickets as Lowe's syndrome and in Wilson's diseases (**sun-flower cataract**).
- 2) **Subluxated lens:** Seen in: Marfan's syndrome and homocystinuria.
- 3) **Corneal rings:** Seen in: Wilson's disease and hypercholesterolemia.
- 4) **Corneal infiltrates and edema:**  
Is a common manifestation of mucopolysaccharidosis and corneal crystals are seen in cystinosis.
- 5) **Lid nodules (xanthomas):** Are a common feature of hyperlipidemia.
- 6) **Cherry-red spot of the fovea:** Is a common finding sphingolipidoses & mucopolipidoses as Tay-Sachs disease and Niemann-Pick disease.
- 7) **Optic atrophy:** Is common in many end-stage metabolic disorders.

## Nutritional Deficiencies

- 1) **Vitamin A deficiency:**  
**Causes:** Xerosis of the Conjunctiva, keratomalacia in severe cases and night blindness.
- 2) **Severe vitamin B deficiency:**  
**As in:** Beriberi and tobacco alcohol amblyopia can produce ophthalmoplegia, various scotomas and finally optic atrophy.
- 3) **Deficiency of vitamin C and other anti-oxidants:**  
**May predispose to:** Cataract and age-related macular degeneration.

# Drug and Chemical intoxication

**1) Blepharo-coniunctivitis:** Is common with many topical and systemic medications causing:

- **Allergy** as with the use of sulphonamides.
- **Darkening of the conjunctiva** (argyrosis) due to prolonged use of topical and systemic silver preparations.
- **Lid and conjunctival pigmentation** can occur with the prolonged use of Latanoprost and epinephrine (anti-glaucoma medications),
- **Bacterial and fungal infections** occur with immune-suppressive therapy.

**2) Corneal deposits:**

- ❖ In a vortex manner **can be seen in** amiodarone therapy and various quinine derivatives.
- ❖ **Copper deposit causes** a blue-green corneal arcus in Wilson's disease (Kayser-Fleisher ring).

**3) Cataract:**

Is a common feature of **long-term corticosteroid therapy**, as well as **open angle glaucoma**.

**4) Closed angle glaucoma:**

Can occur in predisposed eye with the use of drugs containing **atropine derivatives as anti-spasmodics and anti-depressants**.

**5) Changes in the pupil size:**

- ❖ Occur with various drugs affecting the **autonomic nervous system**.
- ❖ Paresis of accommodation is common with **atropine derivatives** while spasm of accommodation occurs with **organo-phosphorus poisons**.

**6) Paresis of the extra-ocular muscles:** Is seen with **chronic lead poisoning** as well as **CNS depressant medications leading to binocular diplopia**.

**7) Retinal toxicity:**

- ❖ Is a feature of chronic therapy with **phenothiazines (pigmentary retinopathy)**, **chloroquines (maculopathy)** and **tamoxifen (used in breast cancer therapy)**.

**8) Papilledema and increased intra-cranial tension:**

Can occur with **chronic therapy with vitamin A, corticosteroids and tetracyclines**.

**9) Optic atrophy:**

Can occur from various intoxications as **methyl alcohol, lead poisoning & anti-tuberculous drugs**.

# Ocular Tumors



- ❖ Tumors may arise from extra-ocular structures such as the lacrimal gland, optic nerve, lids & conjunctiva, or from intra-ocular structures such as the choroid & the retina.
- ❖ The more common **extra-ocular tumors include**: Mixed lacrimal gland tumors, Basal cell carcinoma of the eyelids and Squamous cell carcinoma of the conjunctiva & Glioma of the optic nerve.
- ❖ Benign **intra-ocular tumors** are very rare; the more common malignant tumors may arise from the choroid (**malignant melanoma**) and the retina (**retinoblastoma**).

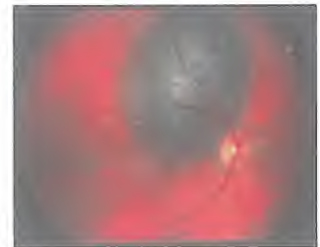
## Intra-Ocular Tumors

### 1) Malignant Melanoma of the Choroid (MM)

- ❖ It is the most common **primary intra-ocular tumor in adults**.
- ❖ The average age of patients with choroidal melanoma is **50 years**.
- ❖ It is rare in **black races**.

#### Clinical features:

- ❖ **The tumor appears as**: unilateral, elevated, brown oval shaped mass.
- ❖ **It may be**: mottled with dark brown & black pigmentation.
- ❖ **Early**: It may be asymptomatic unless it arises near the macular area.
- ❖ **Later**: The patient may complain of diminution of vision **due to**:
  - 1) **Secondary retinal detachment** (malignant detachment).
  - 2) **Secondary glaucoma** (refer to secondary glaucoma in chapter 8).
- ❖ **In late stages**: It may extend outside the globe into the orbit producing proptosis.
- ❖ **Diagnosis is based on**: Fundus examination.
- ❖ Many cases are accidentally discovered during routine ocular examination.
- ❖ **Fundus photography**: important for documentation and follow up.
- ❖ **Ultrasonography**: accurately determines the size & height of the tumor and differentiates MM from other tumors. E.g.: Choroidal nevus.



#### Differential diagnosis:

- ❖ Malignant melanoma **should be differentiated from** a choroidal nevus which is benign.
- ❖ Malignant detachment **should be differentiated from** primary rhegmatogenous retinal detachment.
- ❖ Choroidal melanomas **should be differentiated from** choroidal secondaries due to metastases. Since metastases are frequently bilateral, examination of the fellow eye is extremely important.

## **Management:**

1) **Observation:** should be considered for:

a) Eyes with **small lesions** since differentiation from nevus may be difficult.

b) Single eye with **slowly growing tumors**.

❖ In these cases regular Fundus examination, documents Fundus photographs and Ultrasonography are used to detect the tumor height, size and rate of progression.

2) **Local resection:** for small peripheral lesions.

3) **Radioactive plaques:** fixed to the globe may be suitable for small or medium sized tumors near the posterior pole.

4) **Enucleation:** is indicated for large melanomas.

5) **External beam irradiation:** prior to Enucleation to lessen the risk of metastatic lesions.

6) **Trans-papillary thermotherapy (TTT):**

❖ It is a recent method of treatment.

❖ The principle is to apply heat to the tumor tissue after papillary dilatation using a diode laser.

❖ This leads to cellular destruction and inhibition of DNA synthesis by the tumor cells and subsequently decreases in the tumor size.

## **2) Retinoblastoma**

❖ It is the most common primary intraocular malignancy in childhood.

❖ It occurs in about **1 in 20,000 live births**.

❖ It almost always presents prior to **the age of 3 years**.

❖ A positive family history is present in **only 6% of cases**, while sporadic cases account for the **remaining 94% of cases**.

❖ The tumor may be **bilateral in 20% of the cases**.

❖ It is thought to be caused by a mutation in a gene on **chromosome 13** resulting in the loss of an anti-oncogene.

**Clinical Features:** Retinoblastoma may present as:

1) **Leukocoria:** white papillary reflex, accounts for **65% of cases**.

2) **Strabismus (squint):** if the tumor affects the macular area.

3) **Secondary glaucoma.**

4) **Proptosis** due to extraocular extension.

5) **Accidental discovery on routine examination.**



**Diagnosis:** is based on:

**1) Ophthalmoscopy:**

- ❖ With **maximal papillary dilatation**, preferably under **general anesthesia**.
- ❖ It is of extreme importance to examine both eyes **due to the relatively high incidence of bilaterality**.

**2) X-ray** may show tumor calcification.

**3) Ultrasonography.**

**4) CT scans.**



**Treatment:**

**1) Photocoagulation** for small central tumors.

**2) Trans-scleral cryotherapy** or diode laser for small peripheral tumors.

**3) Radiotherapy** or trans-papillary thermotherapy for medium tumors.

**4) Systemic chemotherapy.**

**5) Enucleation with** excision of a long stump of the optic nerve is the treatment of choice for large tumors affecting one eye.

**Differential Diagnosis Of Leukocoria In Infants:**

**1) Retinoblastoma.**

**2) Congenital cataract.**

**3) Retinopathy of prematurity:** Failure of vascularization of the peripheral retina in prematures exposed to high oxygen tension in incubators with subsequent fibrosis and tractional retinal detachment.

**4) Persistent hyperplastic primary vitreous:** congenital anomaly with fibrosis of the anterior vitreous.

**5) Coat's disease:** unilateral extensive leakage from the retinal vessels resulting in large masses of sub-retinal lipids.

**6) Retinal dysplasia:** chromosomal defect resulting in development of a disorganized retina.



# Extraocular Tumors

## 1) Optic Nerve Glioma

- ❖ This **locally** malignant tumor arises from the **astrocytes** of the **optic nerve** commonly in childhood.
- ❖ It grows **anteriorly** towards the globe **resulting** in proptosis and optic atrophy and **posteriorly** towards the optic chiasma **resulting** in widening of the optic foramen and intra-cranial symptoms.

## 2) Mixed Lacrimal Gland Tumor

- ❖ A **locally malignant tumor of the lacrimal gland** usually affecting middle-aged patients.
- ❖ It is more common in **females with a ratio of 3:2**
- ❖ It **invades** the surrounding structures and extends along the nerve sheaths causing severe pain.

## 3) Secondary Tumors

- ❖ **Orbital metastases** are the most common orbital tumors.
- ❖ In **children**, they may be due to leukemia or neuroblastoma.
- ❖ In **adults**, lymphomas are common as well as secondaries from breast cancer in females and lung cancer in males.

## 4) Phakomatosis

This is a group of diseases characterized by appearance of multiple small benign proliferations, this proliferations affects the eyelid intraocular structures face as well as other organs of the body,

**The most common diseases are:**

- 1) **Neurofibromatosis:** Multiple neurofibromata of the skin and subcutaneous nerves with patches of skin pigmentation. (Café-au-lait patches).
- 2) **Sturge-weber syndrome:** hemangioma affects half of the face and eyelids with severe secondary glaucoma and buphthalmos.
- 3) **Tuberous sclerosis:** Multiple sebaceous adenoma of the face and multiple retinal astrocytoma.
- 4) **Von Hippel-Lindau disease:** Solitary retinal angioma with multiple cysts affecting the cerebellum, pancreas and other organs.

Ocular

pharmacology

### Groups of ocular drugs:

- 1) Mydriatics & cycloplegics,
- 2) Antiglaucoma medications.
- 3) Steroids.
- 4) Anti-infectives.
- 5) Non-steroidal anti-inflammatory drugs.
- 6) Antihistaminics.
- 7) Vasoconstrictors.
- 8) Local anesthetics.
- 9) Tear substitutes.
- 10) Ocular dyes.
- 11) Others: Contact lens solutions, irrigating fluids, astringent drops, mast cell stabilizers, solutions for gonioscopy and laser lenses, and combinations of eye drops are also available.

### N.B. Ocular injections:

#### 1) Intraocular:

a) Intracameral (into the A.C.)

b) Intravitreal (into the vitreous cavity).

#### 2) Periocular:

a) Subconjunctival.

d) Peribulbar.

b) Anterior subtenon.

e) Retrobulbar.

c) Posterior subtenon.

## Mydriatics and Cycloplegics

**Active mydriasis:** by stimulating the dilator pupillae muscle (**sympathomimetics**).

**Passive mydriasis:** by paralysis of the sphincter pupillae muscle (**parasympatholytics**), these also cause paralysis of the ciliary muscle; thus inhibiting accommodation (**cycloplegia**).

### **Active mydriatics (sympathomimetics)**

a) Phenylephrine hydrochloride: 2.5% & 10% eye drops.

➤ Onset: 20 minutes.

➤ Duration: 4 hours.

b) Adrenaline: 2-5% ampoules.

➤ Given 1/1000 by subconjunctival injection or 1/100,000 by intracameral injection to be added to the irrigating solution **as during**: Cataract extraction.

### **Passive mydriatics (parasympatholytics)**

Drug	Mydriasis		Cycloplegic effect
	Peak (min)	Recovery (days)	
Atropine 0.5-1 %	30-40	7-10	++++
Cyclopentolate 0.5-2 %	30-60	1	++
Tropicamide 0.25-1 %	20-40	0.25	+



## **Uses of mydriatics:**

### **1) Diagnostic:**

- a) **Before retinoscopy:** in children and young adults, good cycloplegia is necessary.
- b) **Before fundus examination:** a short acting cycloplegic is used as Tropicamide.
- c) **To measure vision:** after mydriasis in the presence of central opacities (mydriatic test).

### **2) Therapeutic:**

- a) **In keratitis and corneal ulcers:** short acting cycloplegics are given. A strong cycloplegic is required in **resistant ulcers**.
- b) **In Iridocyclitis:** a strong cycloplegic is necessary e.g. **Atropine**.
- c) **In strabismus:** Treatment of accommodative esotropia in children **less than 2 years**.
  - As a method of **treating amblyopia**, cycloplegics are used in the seeing Eye, to force the patient to use his amblyopic eye.

### **3) In surgical procedures:**

- a) **Pre-operative:** To dilate the pupil **before** cataract and retinal detachment surgery.
- b) **Operative:** To induce pupillary dilatation (**adrenaline added to solutions**) during surgery in cases where the pupil becomes constricted during the operation.
- c) **Post-operative:** A **short acting** cycloplegic is used in cataract, retinal detachment and glaucoma surgeries.

## **Dangers of mydriatics:**

1) **Angle closure glaucoma** in patients with a narrow angle.

2) Prevents monitoring of the pupil in **patients with concussion & during anesthesia**.

### **3) Atropine toxicity (over dose):**

- Toxicity is **due to** systemic absorption.
- Atropine ointment is thus indicated to **decrease** the possibility of systemic absorption.
- **Manifestations:**
  - ❖ Fever.
  - ❖ Flushing of the face,
  - ❖ Tachycardia.
  - ❖ Fits (CNS excitement).
  - ❖ Dry mouth & skin.
- **Treatment:** stop atropine, cold compresses, **pilocarpine 10 mg IM (anti-dote)**.

### **4) Atropine sensitivity (allergic reaction to atropine):**

- This may occur at any age and may occur with drops or ointment.
- **Manifestations:** allergic dermatitis and follicular conjunctivitis.
- **Treatment:** stop atropine, give cyclopentolate instead and give local steroids.

5) **Prolonged use of atropine may lead to dry eye.**

# Anti-Glaucoma Medications

(See glaucoma) +

## Uses of miotics:

### 1) Antiglaucoma medications:

- a) **Angle closure glaucoma** during the acute attack and prophylactic in the other eye.
- b) **Open angle glaucoma** in conjunction with beta blockers and carbonic anhydrase inhibitors in resistant cases.

### 2) Other uses of miotics:

#### a) **Preoperative:**

- ❖ Before glaucoma surgery.
- ❖ Penetrating keratoplasty.
- ❖ Goniotomy.
- ❖ Laser iridotomy.

#### b) **Intraoperative:**

- ❖ Goniotomy.
- ❖ Cyclodialysis.
- ❖ Cataract extraction to constrict the pupil at the end of surgery.

#### c) **Some cases of accommodative squint (long acting miotics are used).**

## Side effects of miotics:

1) **Ocular:** constriction of the field of vision, worsening of vision in patients with nuclear cataract, spasm of accommodation, and may cause cataract. Excessive use may cause pupillary block glaucoma.

2) **Systemic:** bradycardia, gastrointestinal colics, headache.

# Corticosteroids

## Types:

- 1) Hydrocortisone.
- 2) Prednisone, prednisolone (4 times stronger than hydrocortisone) e.g. Prednisolone acetate 1%.
- 3) Dexamethasone (25 times stronger). E.g. Dexamethasone phosphate.
- 4) Fluometholone acetate 0.1% (less possibility of increasing the IOP).

### Forms:

- ❖ **Topical:** drops and ointment.
- ❖ **Vials:** for injection or infusion.
- ❖ **Tablets:** for oral use.

### Indications:

- 1) **Lids:** allergic dermatitis and insect bites.
- 2) **Conjunctiva:** allergic conjunctivitis (phlyctenular & spring catarrh).
- 3) **Anterior segment:**

- a) Keratitis (interstitial & deep).
- b) Episcleritis & scleritis.
- c) Iridocyclitis.
- d) Acute phase (1st 10 days) of chemical injuries & burns.

**N.B. All the above corticosteroids are used topically or by local injections.**

- 4) **Posterior segment:** In these conditions topical steroids are not effective and either systemic or retro-bulbar injections are used.
  - a) **Anterior ischemic optic neuropathy:** where mega doses of steroids may be used. The patient is hospitalized and doses as high as one gram are given intravenously under the supervision of an internist.
  - b) **Posterior Uveitis.**
  - c) **Sympathetic ophthalmitis.**
- 5) **Post-operative:** to decrease post-operative inflammation and also to prevent graft rejection in cases of keratoplasty.

### Dangers of local steroids:

- 1) Steroid induced glaucoma.
- 2) Steroid induced cataract (post sub-capsular).
- 3) Increasing susceptibility to infections.
- 4) Reactivation of dormant organisms e.g. Herpes simplex.
- 5) Delaying wound healing.

### Dangers of systemic steroids (prolonged use):

- 1) Peptic ulceration.
- 2) Steroid induced diabetes.
- 3) Hypertension due to salt and water retention.
- 4) Steroid induced cataract.
- 5) Muscle wasting & osteoporosis.



- 6) Reactivation of dormant infections (TB.).
- 7) Cushingoid state.
- 8) Psychic disturbance.
- 9) Sudden stoppage after prolonged use may lead to acute adrenal insufficiency, so either gradual withdrawal or injection of ACTH is given at the end of treatment.

## Local Anti-Infective Drugs

### 1) Anti-bacterial:

Drops	Ointments
Sulfacetamide 10-30%	Erythromycin 0.5%
Chloramphenicol 0.5%	Chloramphenicol 1.0%
Gentamycin 0.3%	Gentamycin 0.3%
Tobramycin 0.3%	Tobramycin 0.3%
Ciprofloxacin 0.3%	Oxytetracyclin 0.5% (Terramycin)
Ofloxacin 0.3%	-----

**N.B.:** All of the above mentioned medications are bactericidal.

Except sulfacetamide which is bacteriostatic

### 2) Anti-viral: (See cornea).

### 3) Anti-fungal (anti-mycotic): (See cornea).

## Non-Steroidal Anti Inflammatory Drugs

### Types:

#### 1) Local:

- a) Diclofenac 0.1% drops (Voltaren).
- b) Ketorolac 0.5% drops (Acular).

#### 2) Systemic: as Diclofenac 50 mg t.d.s.

### Uses:

- 1) In anterior segment inflammatory disease.
- 2) In mild cases of Uveitis.
- 3) When there are contraindications to local steroids.
- 4) Preoperative & postoperative.
- 5) CME (cystoid macular edema).

# Ocular Dyes

## 1) Fluorescein sodium:

- It is a yellow water soluble dye.
- Doesn't stain cells or mucous.

### **Uses:**

- 1) **Demonstrate** defects in the corneal epithelium i.e diagnosis of corneal ulcers.
- 2) Applanation tonometry.
- 3) Rigid contact lens **fitting**.
- 4) Tear break up time **determination**.
- 5) Lacrimal drainage system **evaluation**.
- 6) **Siedel's test** (for wound leak and corneal fistula).
- 7) **Fluorescein angiography: 10-25% IV.**

### **Indications:**

- Diagnosis and management of **vaso-occlusive diseases**,
- Diagnosis and management of **retinopathies**.
- Diagnosis and management of **macular diseases**.

## 2) Rose Bengal:

- An iodine derivative of fluorescein.
- It stains corneal & conjunctival cells (including the nuclei and cell walls) by a red color.

## 3) Indocyanin green:

- Used to visualize the choroidal circulation by infrared angiography.

# Laser In Ophthalmology

**Definition of Laser:** Light Amplification by Stimulated Emission of Radiation (LASER).

## **Classification of Lasers:**

### According to the wave length:

- 1) **Visible Lasers:** Lying in the visible portion of the spectrum, e.g.: Argon laser with a wavelength 488-514 nm 'coagulating laser'.
- 2) **Invisible Lasers:** Lying either in the ultraviolet portion of the spectrum (e.g.: excimer laser with a wave length 193 nm 'ablating laser') or in the infrared portion (e.g.: carbon dioxide laser with a wave length and YAG laser with a wavelength 1064 nm 'cutting lasers').

### According to the state of Laser:

- ❖ **Solid state lasers:** as YAG laser.
- ❖ **Liquid state lasers:** as dye lasers.
- ❖ **Gas state lasers:** as excimer and CO<sub>2</sub> lasers.

## **Uses of LASER in ophthalmology:**

- 1) **Lid disorders:** Removal of warts and papillomas. (CO<sub>2</sub> laser is used).
- 2) **Errors of refraction:** Excimer laser photo-ablation (LASIK and PRK) are widely used to correct a wide range of refractive errors.
- 3) **Cataract:**
  - ❖ YAG laser capsulotomy to treat late postoperative posterior capsule **opacification** following cataract surgery.
  - ❖ **Erbium-YAG laser:** to remove the cataractous lens.
- 4) **Glaucoma:**
  - ❖ Laser iridotomy using YAG laser.
  - ❖ Argon or diode laser trabeculoplasty in some cases of open angle glaucoma.
  - ❖ Scanning laser Ophthalmoscopy to visualize the optic nerve head in cases of glaucoma (**see glaucoma**).
- 5) **Retinal disorders:**
  - ❖ Argon or diode laser may be used to seal retinal tears, as well as in the treatment of diabetic retinopathy and retinal vascular disorders.

## **Complications of Laser:**

- 1) **Opacification:** of cornea, lens or vitreous.
- 2) **Hemorrhage:** Hyphema, Vit. Hemorrhage & retinal hemorrhage.
- 3) **Foveal damage** in uncooperative patients.
- 4) **Increased I.O.P** (Transient).



# Differential Diagnosis

## 1) Acute red eye:

- ❖ Conjunctivitis
- ❖ Corneal ulcer
- ❖ I.C.
- ❖ Acute congestive glaucoma
- ❖ Orbital cellulitis
- ❖ C.S.T.
- ❖ Endophthalmitis
- ❖ Panophthalmitis
- ❖ Scleritis & Epi-scleritis
- ❖ Sub-conjunctival hge

## 2) Diabetes & the eye: See part 2.

## 3) Causes of Leukocoria: See congenital cataract.

## 4) Causes of cherry red spot:

- ❖ CRAO
- ❖ Commotio Retinae
- ❖ Quinine poisoning
- ❖ Macular hole
- ❖ Tay sachis disease (mucopolipidosis)

## 5) Causes of sudden loss of vision:

- ❖ CRAO
- ❖ Rupture globe (traumatic)
- ❖ Amaurosis fugax
- ❖ Hysterical

## 6) Causes of gradual ↓ V/A:

- ❖ Senile cataract
- ❖ OAG
- ❖ Malignant myopia
- ❖ AMD (age related macular degeneration)
- ❖ Chronic I.C.
- ❖ P.O.A
- ❖ Retinitis pigmentosa
- ❖ Keratoconus

## 7) Causes of rapid diminution of vision:

### a) Hours:

- ❖ Acute congestive glaucoma (painful)
- ❖ Commotio Retinae (traumatic)
- ❖ Vitreous hge (painless)
- ❖ CRVO (painless)

### b) Days:

- ❖ Corneal ulcer
- ❖ I.C.
- ❖ Choroiditis
- ❖ R.D.
- ❖ Optic neuritis

### 8) Night blindness:

- ❖ Retinitis pigmentosa
- ❖ Vit. A deficiency
- ❖ Sedrosis bulbi (pseudo retinitis pigmentosa)
- ❖ Cortical cataract
- ❖ Cong. Night blindness
- ❖ OAG (advanced)

### 9) Day blindness:

- ❖ Central corneal opacity
- ❖ Nuclear cataract

### 10) Diplopia:

#### a) Uni-ocular:

- ❖ Early cortical cataract
- ❖ Subluxated lens
- ❖ Irido-dialysis
- ❖ Anisometropia (corrected by glasses)

#### b) Binocular:

- ❖ Paralytic squint
- ❖ Decompensated latent
- ❖ Proptosis (unequal) (unilateral)
- ❖ Symblepharon & Ptregium
- ❖ Displaced globe (by orbital tumors)
- ❖ Anisometropia (if corrected by glasses)

### 11) Causes of Miosis & Mydriasis: See part 2 (Neuro).

### 12) Color blindness (Achromatopsia):

#### a) Congenital (common) (more in males):

- ❖ Total (black & white only)
- ❖ Partial (one of the 3 primary colors is lost blue, red & green)

#### b) Acquired:

- ❖ Papillitis (2 primary colors are lost red & green)
- ❖ Papilledema (Only 1 primary color is lost, blue)

**N.B.** ➤ Tested by:

- 1) Ishihara chart.
- 2) Colored beads.
- 3) Mature cataract (colored light).

**13) Photophobia:**

- ❖ Congenital glaucoma
- ❖ Iridocyclitis
- ❖ Acute angle closure glaucoma
- ❖ Foreign body (corneal or conjunctival)
- ❖ Corneal abrasion
- ❖ Keratitis

**14) Vitreous floaters:**

- ❖ Vitreous degenerations
- ❖ Vitreous hemorrhage
- ❖ Posterior Uveitis
- ❖ Vitreous detachment

**15) Swollen optic disc (disc edema):**

**a) Bilateral:**

- ❖ Papilledema
- ❖ Anterior ischemic optic neuropathy
- ❖ Systemic infections (TB and syphilis)
- ❖ Benign increased intracranial tension
- ❖ Meningitis

**b) Unilateral:**

- ❖ Diabetic retinopathy
- ❖ Central retinal vein occlusion
- ❖ Ocular ischemic syndrome
- ❖ Hypertensive retinopathy
- ❖ Optic neuritis.



## 16) Abnormalities of the anterior chamber:

### a) Abnormal depth:

- Shallow anterior depth:
  - ❖ **Short axial length:** Hypermetropia and microphthalmos.
  - ❖ Acute angle closure glaucoma
  - ❖ **Pupillary block:** intumescent cataract, anterior dislocation of the lens and occlusion of pupillae.
- Deep anterior chamber:
  - ❖ Aphakia
  - ❖ Hypermature cataract
  - ❖ Posterior dislocation of the lens
  - ❖ High myopia
  - ❖ Buphthalmos
- Irregular depth:
  - ❖ Leucoma adherent
  - ❖ Subluxation of the lens

### b) Abnormal contents:

- ❖ Blood (hyphema)
- ❖ Inflammatory cells
- ❖ Dislocated lens
- ❖ Excessive proteins (Plasmoid aqueous)
- ❖ Pus (hypopyon)
- ❖ Foreign body



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# **AVAILABLE BOOKS**

**PART 1**

**PART 2**

**MCQ BANK**

**ATLAS OF OPHTHALMOLOGY**

University Book Centre  
Sayed Mahmoud  
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